Appendix A to RI Report

Endangerment Assessment

of Middle Fork of Little Beaver Creek

Nease Chemical Company – Salem, Ohio Site

Volume 2 of 4

Submitted to

United States
Environmental Protection Agency
Region 5, Chicago, Illinois

and

Ohio Environmental Protection Agency
Columbus, Ohio

April 5, 1991

Submitted by

Ruetgers-Nease
Chemical Company, Inc.
State College, Pennsylvania

ENDANGERMENT ASSESSMENT FOR THE NEASE CHEMICAL COMPANY SALEM, OHIO SITE MIDDLE FORK OF LITTLE BEAVER CREEK

Prepared for

Ruetgers-Nease Chemical Company, Inc.
State College, PA

Prepared by

ENVIRON® Corporation
Arlington, Virginia

April 4, 1991

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I. INTRODUCTION

A. Background

Ruetgers-Nease Chemical Company, Inc. (Ruetgers-Nease) owns an inactive manufacturing site in Salem, Ohio (Site). In 1983, the Site was placed on the National Priorities List (NPL). A number of studies have been conducted to characterize the Site and the surrounding area. Most recently, ERM-Midwest, Inc. completed a sampling program for the Middle Fork of Little Beaver Creek (MFLBC), the principal surface water body receiving runoff from the Site, as part of the Remedial Investigation (RI). This sampling program included the collection of surface water, stream sediment, flood plain soils, and fish tissue samples from locations along the MFLBC from upstream of the Site to near East Liverpool, Ohio. Sampling was conducted from April 16 to May 21, 1990. A full description of the sampling program and its results is presented in the RI for the MFLBC prepared by ERM-Midwest, Inc. (1991).

ENVIRON Corporation (ENVIRON) was retained by Ruetgers-Nease to prepare an Endangerment Assessment (EA) for the MFLBC. The objective of the EA, also referred to as the baseline risk assessment, is to characterize the potential risks to public health and the environment associated with hazardous substances that may have migrated from the Site to the MFLBC in the absence of any further remediation or institutional controls (i.e., under an assumption of no action). Consistent with this guidance, ENVIRON has considered in the EA only those chemicals that are potentially site-related.

B. The Risk Assessment Process

The assessment of potential risks described in this document is based on guidelines provided by the U.S. Environmental Protection Agency (USEPA) and is consistent with the steps of the risk assessment process as described by the National Academy of Sciences (NAS) in a report prepared by its Committee on the Institutional Means for Assessment of Risk to the Public Health (NRC 1983). The guidelines and methodology described by USEPA and NAS have been established for the assessment of risks posed by environmental agents in a regulatory context. As emphasized by the Office of Science and Technology Policy (OSTP 1985) and USEPA (1986a) with respect to carcinogenic risk assessments, these assessments involve a number of assumptions and forms of extrapolation that have not been verified by traditional scientific means. This approach has arisen because of the need, as perceived by

regulatory officials, to act in the absence of complete experimental information by adopting a series of conservative and often unproven assumptions to ensure maximum health protection. Risk assessments performed in this manner are designed to place an upper bound on risk (USEPA 1986a). Similarly, risk assessment methods developed for the noncarcinogenic effects of chemicals incorporate various conservative (i.e., health protective) assumptions. Noncarcinogenic risk assessment is not intended to provide a demarcation between "safe" and "unsafe" levels of exposure. A substantial margin of safety is built into noncarcinogenic toxicity values[1], thereby providing a high degree of certainty that the levels derived as "acceptable" according to methods developed by regulatory agencies will cause no adverse health effects in the potentially exposed population. Exposures may even exceed the acceptable dose level without a significant risk arising.

It must be emphasized that the risks estimated using these risk assessment methods are not actuarial, i.e., the risk estimates cannot be used to predict the actual number of individuals who might experience health consequences as a result of exposure. Actual health risk is almost certainly less than that described using the methods of risk assessment. Furthermore, the risk estimates developed herein do not relate to individual risks. Many individual risk factors -- such as exposures to other environmental agents, occupational exposures, smoking, age, diet, and inherent susceptibility -- will influence the probability of developing a specific disease.

Although current risk assessment approaches generally overstate risk, they nevertheless provide a systematic approach that allows public health policymakers to establish the relative risk posed by various environmental substances and potential exposure pathways. A further discussion of uncertainties in the risk assessment process and the conservative assumptions adopted in light of this uncertainty is presented in Chapter IV, Toxicological Assessment (see pp. 23 - 24 for a more detailed discussion of uncertainties in the development of toxicity values) and Chapter VIII, Risk Characterization (see pp. 64-66 and 75-80 for a more detailed discussion of uncertainties in risk assessment).

[1] Noncarcinogenic toxicity values are referred to by USEPA as reference doses (RfDs). The term RfD is further described in Chapter IV, Toxicological Assessment.

C. Report Organization

This report, which has been prepared in accordance with USEPA risk assessment guidance (USEPA 1989a, 1989b), is divided into ten chapters as follows:

Chapter 1, Introduction, in which background on the project, a discussion of the risk assessment process, and the report organization are presented.

Chapter 2, Site Description and History, in which the description and history of the Site relevant to the assessment of human health and environmental risks associated with the MFLBC are summarized.

Chapter 3, Identification of Chemicals of Potential Concern, in which chemicals of potential concern are selected to focus the assessment on those site-related chemicals that are most likely to pose the greatest potential public health risk.

Chapter 4, Toxicological Assessment, in which the hazard identification and dose-response evaluation for each chemical of potential concern -- mirex and photomirex -- are accomplished to derive toxicity values that can be used to estimate the potential for adverse effects occurring in humans at different exposure levels.

Chapter 5, Identification of Exposure Pathways, in which potential exposure pathways under current and hypothetical future conditions of the area along the MFLBC are identified.

Chapter 6, Estimation of Environmental Concentrations, in which chemical concentrations are estimated for the various environmental media associated with the potential exposure pathways.

Chapter 7, Estimation of Human Intake, in which estimates of chemical concentrations at the points of human exposure are combined with exposure assumptions (e.g., the duration of exposure, the amount of chemical absorbed in the body, and the characteristics of the population receiving the exposure) to arrive at estimates of human intake or dose.

Chapter 8. Risk Characterization, in which numerical estimates of carcinogenic and noncarcinogenic risks are calculated for each chemical by each potential route of exposure using the toxicity information and the estimates of human intake.

Chapter 9. Comparison of Environmental Concentrations of Chemicals of Potential Concern to Applicable or Relevant and Appropriate Requirements (ARARs), in which potential chemical-specific ARARs are identified and compared to the chemical concentrations found or modeled at the MFLBC.

Chapter 10. Environmental Risk Assessment, in which the principles of risk assessment are used to evaluate the potential effects on local flora and fauna.

In addition, a number of technical appendices to the report provide the necessary documentation of data and methods relied upon to perform the analyses.

The environmental data contained in this EA are based solely on surface water, sediment, soils, and fish tissue sampling results presented in the RI for the MFLBC (ERM-Midwest, Inc. 1991), and on residue data for game collected by the Ohio Department of Health (ODH 1990).

II. SITE DESCRIPTION AND HISTORY

A. Introduction

This chapter presents a summary of the description and history of the Site relevant to the assessment of human health and environmental risks associated with the MFLBC. A more detailed description and history of the Site is presented in the RI (ERM-Midwest, Inc. 1991).

B. Site Description

The Site is located in northeastern Ohio in a rural area northwest of the City of Salem. The Site, consisting of approximately 44 acres, is surrounded by residential and farm land on three sides with an industrial plant to the northeast. The MFLBC, located less than 1500 feet from the Site, is the principal surface water body receiving runoff from the Site. The MFLBC originates near the Site in Salem and is connected with the property via Feeder Creek. From Salem, the MFLBC flows north for about five miles, then turns and flows southeastward through Lisbon, Ohio where it eventually joins other tributaries to become Little Beaver Creek. Little Beaver Creek flows into the Ohio River near East Liverpool, Ohio.

C. Site History

From January 1961 until December 1973, a chemical manufacturing plant operated at the Site. During this period, Nease Chemical Company (Nease), which owned the Site, manufactured a variety of specialty chemicals including household cleaning compounds, fire retardants, pesticides, and chemical intermediates used in agricultural, pharmaceutical, and other chemical products. Products and chemical intermediates were manufactured in batch processes, and finished products were stored in warehouses, bulk storage, and tanks. Wastes generated during the production of chemicals were neutralized and treated in five on-site ponds. Effluent from the ponds was discharged to the Salem Wastewater Treatment Plant from the late 1960s to 1975. A complete list of products and raw materials as provided by Ruetgers-Nease is included in Table 1.

Manufacturing operations at the Site were discontinued in 1973. All of the buildings and manufacturing equipment on the Site, except for a warehouse and two small block buildings, were removed during decommissioning activities.

TABLE 1
Chemical Compounds Used and Produced at the Facility

Raw Materials

*Acetic Acid
Acetone
Aluminum Chloride
Ammonium Chloride
Anisole
*Aqua Ammonia
Barium Hydroxide
Benzaldehyde
*Benzene
Benzoic Acid
Benzoyl Chloride
*Bromine
Calcium Chloride
Carbon Tetrachloride
*Caustic Soda
Celite
Celkate
Chloral
*Chlorine
Chlorobenzene
Chloroform
p-Chlorophenol
*Chlorosulfonic Acid
Dibromopropanol
*o-Dichlorobenzene
3,4-Dichloronitrobenzene
Epichlorohydrin
Ethyl Chloroformate
Ethylene Dichloride
Ethylene Glycol
Fibra Flo
Filtrol
Formaldehyde
Glycine
Heptane
Hexachlorocyclopentadiene
Hydrochloric Acid (aqueous)
Iodine
Iron
*Isopropyl Alcohol
Magnesium Oxide
Melamine
*Methanol
Methyl Isocyanate
Naphthalene
*Nitric Acid
m-Nitrobenzotrifluoride
Nuchar
Oleum
o-Phenylphenol
Phosphorus Oxychloride
o-Phenylphenol
Phosphorus Oxychloride
Potassium Persulfate
Soda Ash
Sodium Chloride
Sodium Cyanide
*Sodium Hypochlorite
Sodium Isopropyl Xanthate
Styrene
*Sulfuric Acid
1,1,2,2-Tetrachloroethane
*1,1,2,2-Tetrachloroethene
Tetrine Acid
*Toluene
Trichloroethene
Urea
Xylene
Zinc

Products

m-Amino Benzotrifluoride
Benzene Sulfonamide
Benzene Sulfonic Acid
*Benzene Sulfonyl Chloride (BSC)
p-Chlorobenzene Sulfonamide

TABLE 1
(Continued)

Products (cont'd)
p-Chlorobenzene Sulfonic Acid
p-Chlorobenzene Sulfonyl Chloride
5-Chloro-2-Hydroxybenzophenone
4,4'-Dibromobenzil
Dichlorophene
2-(3,4-Dichlorophenyl)-1,2,4-
Oxadiazoline-3,5-Dione (Compound 438)
Diisopropyl Dixanthogen (DIP)
Diphenyl Sulfone (DPS)
Ethylene Glycol Dibenzene Sulfonate (EGDBS)
Hexabromobenzene
Hexachloroethane
Hippuric Acid
Hydrochloric Acid
Methoxychlor
β-Naphthalene Sulfonic acid (34% Solution)
p-Nitroacetophenone
Perchloropentacyclodecane (Mirex)
Sodium Benzene Sulfochloramide (Chloramine B)
o,p-Toluene Sulfonamide
o,p-Toluene Sulfonyl Chloride
Trichloromelamine
Tris-(2,3-Dibromopropyl) Phosphate
3-(2-Xenoxy)1,2-Epoxypropane

Footnote

*Raw materials and products that were stored in bulk on-site.

As of December 30, 1977, Nease's assets (including the vacant Site) were acquired and merged with Ruetgers Chemicals, Inc. The new company resulting from the merger was Ruetgers-Nease Chemical Company, Inc.

Environmental investigations at the Site and surrounding area began in 1982 at the request of the Ohio Environmental Protection Agency (OEPA). In September 1983, the property was placed on the NPL. In samples of fish and sediment taken by OEPA and USEPA in 1987 and 1988, the Agencies reported the presence of mirex beginning in the MFLBC in the vicinity of the Site property and extending downstream to the dam at Lisbon. Sediments below the dam and downstream to the Ohio River did not contain detectable concentrations of mirex. The fish tissue and sediment data prompted the Ohio Department of Health (ODH) in 1987 to issue a fish consumption advisory and in 1988 to expand the advisory to include a warning against wading and swimming in the stretch of the creek from Salem (junction of the MFLBC and Alternate Route 14) downstream to near the Lisbon Dam (junction of the MFLBC and Route 11). Detailed warning signs were put in place along the affected portions of the creek during the summer of 1988. In 1989, Ruetgers-Nease installed fences on two farms to prevent access of livestock to the MFLBC. Bridges also were constructed on the farms to allow livestock to cross the creek.

The most recent MFLBC sampling program was conducted by ERM-Midwest, Inc. and included the collection of surface water, stream sediment, flood plain soils, and fish tissue samples from locations along the MFLBC from upstream of the Site to near East Liverpool, Ohio. Sampling was conducted from April 16 to May 21, 1990. A description of the sampling program and its results is presented in the RI (ERM-Midwest, Inc. 1991). In addition, data for mirex residues in wildlife tissues obtained by the ODH in September and October 1989 are presented in Appendix A.

III. IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN[2]

A. Introduction

In the RI, 22 chemicals detected in the surface water, sediment, flood plain soil, or fish at the MFLBC were identified as being potentially Site-related (i.e., chemicals that have been identified at the Site as well as in MFLBC samples and therefore may or may not have been Site derived). Tables 2 through 5 present summary statistics, including frequency of detection, maximum and minimum detected levels, and the sample quantitation limits, for each of the potentially Site-related chemicals detected in these media.

Many of the potentially Site-related chemicals are unlikely to contribute significantly to the overall public health risk because of low detected concentrations or low intrinsic toxicity. Therefore, in order to avoid the unnecessary effort of calculating risks for all potentially Site-related chemicals, those chemicals that may pose the greatest potential public health risk are identified. These chemicals of potential concern are then carried through the remaining steps of the baseline risk assessment.

B. Evaluation of Concentration and Toxicity

1. Methodology

The methodology used in the selection of chemicals of potential concern is consistent with guidance provided by the USEPA (1989a), which states that important factors for ranking chemicals of potential concern are their measured concentrations and toxicity. USEPA recommends that for each medium a score based on toxicity and concentration be developed for each compound using the formula:

$$R_{ij} = (C_{ij})(T_{ij})$$

where

[2]Chemicals of potential concern are defined by USEPA (1989a) as chemicals that are potentially Site-related and whose data are of sufficient quality for use in the quantitative risk assessment.

TABLE 2
Potentially Site-Related Chemicals Detected in Surface Water

Chemicals	Frequency of Detection[1]	Range of Sample Quantitation Limits (µg/kg)	Range of Detected Concentrations (µg/kg)
Chloromethane	2/8	10	3.0J-4.0J
Diphenyl Sulfone	1/22	10	2.0J

Footnote

[1] The frequency of detection indicates the number of samples in which the compound was identified or estimated over the total number of samples analyzed.

J Indicates that the quantitation is approximate due to limitations identified during the quality control review.

TABLE 3
Potentially Site-Related Chemicals Detected in Sediment

Chemical	Frequency of Detection[1]	Range of Sample Quantitation Limits (μg/kg)	Range of Detected Concentrations (μg/kg)
Acetone	4/6	11-13	27J-80J
Anthracene	1/24	432.3	340J
Benzoic Acid	2/24	363-396	210J-430J
2-Butanone	1/6	11-13	10J
1,2-Dichloroethane	1/7	11-13	2J
Di-n-butylphthalate	3/16	363-419	43J-74J
Diphenyl Sulfone	2/31	369.6-402.6	55J-170J
Fluoranthene	18/24	349.8-551.1	46J-1100
Mirex	34/52	18.5-1396.8	4.3-1680
2-Methylnaphthalene	4/24	379.5-478.5	46J-100J
4-Methylphenol	6/24	363-419	230J-2800
Naphthalene	3/24	409-479	51J-140J
Phenol	2/24	399.3-419	120J-160J
Photomirex	7/52	25.1-34.3	0.479J-7.38J

Footnotes

[1] The frequency of detection indicates the number of samples in which the compound was identified or estimated over the total number of samples analyzed. Background samples (#1, #29, #30, #47 and #50) were not included for purposes of determining frequency of detection.

J Indicates that the quantitation is approximate due to limitations identified during the quality control review.

TABLE 4
Potentially Site-Related Chemicals Detected in Flood Plain Soil

Chemical	Frequency of Detection[1]	Range of Sample Quantitation Limits (μg/kg)	Range of Detected Concentrations (μg/kg)
Mirex	18/28	21.4-3402.8	16.4J-4540
Photomirex	11/28	22.6-3960	2.5J-132J

Footnotes

1 The frequency of detection indicates the number of samples in which the compound was identified or estimated over the total number of samples analyzed.

J Indicates that the quantitation is approximate due to limitations identified during the quality control review.

TABLE 5
Potentially Site-Related Chemicals Detected in Fish

Chemical	Frequency of Detection[1]	Range of Sample Quantitation Limits (µg/kg)	Range of Detected Concentrations (µg/kg)
Acetone	5/7	12.4-118	120-820J
Benzene	1/7	59	2J
Benzoic acid	2/18	1996.5-48,510	58J-3,300J
2-Butanone	3/7	11.8-100.1	90J-57
Butylbenzyl phthalate	1/48	399.3	360J
Di-n-butylphthalate	4/47	409.2-8184	50J-1400J
Dimethylphthalate	1/43	2,013	210J
Ethylbenzene	3/7	6.05-59	4.0J-5.0J
Methylene chloride	2/7	5.9-6.2	19J-310
Mirex	53/56	17.14-4485.6	5.2J-6150
Phenol	2/10	818.4-2013	93J-380J
Photomirex	36/55	10.97-162.2	1.55J-390J
Tetrachloroethene	1/7	50.5	7J
Toluene	2/7	6.05-59	11J-16J
Xylene (total)	1/7	59	20J

Footnotes

[1] The frequency of detection indicates the number of samples in which the compound was identified or estimated over the total number of samples analyzed.

J Indicates that the quantitation is approximate due to limitations identified during the quality control review.

R_{ij} = risk factor for chemical i in medium j;

C_{ij} = maximum concentration of chemical i in medium j; and

T_{ij} = toxicity value for chemical i in medium j. The slope factor (SF) is used for carcinogens; the inverse of the Reference Dose (RfD) is used for noncarcinogens. These terms are described in greater detail in Chapter IV, Toxicological Assessment.

The inputs to the above equation, media-specific maximum concentrations and USEPA-published toxicity values for each chemical, are derived from data presented in Tables 2 through 6. USEPA guidance (1989a) recommends that when toxicity values are available for only a portion of a class of compounds, consideration be given to treating them as a group in the selection of chemicals of potential concern. At the MFLBC, polycyclic aromatic hydrocarbons (PAHs) were treated in this way. The concentrations of the noncarcinogenic PAHs that were identified as being potentially Site-related (viz., anthracene, 2-methylnaphthalene, and naphthalene) were summed and the total noncarcinogenic PAHs were conservatively assumed to have an RfD equal to that of their most toxic member, naphthalene.

Chemicals w/out Toxicity values cannot be screened using this procedure See RAGS 5-24

After calculation of individual R_{ij} values for each chemical, a total R_j for each medium is calculated as follows:

$$R_j = R_{1j} + R_{2j} + R_{3j} + \ldots + R_{ij}$$

where:

R_j = total risk factor for medium j; and

$R_{1j} + \ldots + R_{ij}$ = risk factors for chemicals 1 through i in medium j.

Chemicals of potential concern are selected based on their relative contribution to the total risk factor for that medium, which is represented by the ratio of R_{ij}/R_j. USEPA (1989a) suggests that chemicals with risk factor ratios above one percent be selected

TABLE 6
Toxicity Parameters for Potentially Site-Related Chemicals

Chemical	Oral Reference Doses (mg/kg/day)[a]	Oral Slope Factors (1/mg/kg/day)[a]	Weight of Evidence[b]
Acetone	0.1		
Anthracene	0.3		
Benzene		0.029	A
Benzoic Acid	4		
2-Butanone	0.05		
Butylbenzylphthalate	0.2		
Chloromethane		0.013	B2
Dichloroethane (1,2-)		0.091	B2
Diphenyl Sulfone			
Di(n)butylphthalate	0.1		
Dimethylphthalate	1		
Ethylbenzene	0.1		
Methylene Chloride	0.06	0.0075	B2
Methylphenol (4-)	0.05		
Mirex	0.000002	1.8	B2
[redacted]			
Phenol	0.6		
Photomirex			
Tetrachloroethene	0.01	0.051	B2
Toluene	0.2		
Xylene (total)	2		

[a] With one exception, all RfDs and Slope Factors are taken from Health Effects Assessment Summary Tables: Fourth Quarter 1990 or the Integrated Risk Information System (IRIS), USEPA Office of Health and Environmental Assessment, Cincinnati, OH. Evaluation of toxicity parameters for noncarcinogenic PAHs is discussed in Chapter III.

[b] USEPA's weight-of-evidence classification system for carcinogenicity:
A = human carcinogen; B2 = probable human carcinogen (USEPA 1989a).

for consideration as chemicals of potential concern. Chemicals that score above the cutoff for any of the media evaluated are retained for further consideration as chemicals of potential concern (i.e., a chemical must score below the cutoff for all media to be eliminated from consideration as a chemical of potential concern).

2. Results of Scoring Chemicals

Only two potentially Site-related chemicals (viz., chloromethane and diphenyl sulfone) were detected in surface water. Similarly, only two potentially Site-related chemicals (viz., mirex and photomirex) were detected in the flood plain soil. Because of the small number of chemicals detected in these media, R_{ij} values were not calculated in this step.

Tables 7 through 10 present the results of scoring the potentially Site-related chemicals for the sediment and fish media. As can be seen in the tables,

- Eighteen potentially Site-related chemicals scored below the established cutoff of one percent of the noncarcinogenic or carcinogenic score in all media, and therefore were eliminated from further consideration as chemicals of potential concern.

- Mirex is the only potentially Site-related chemical scoring above the established cutoff of one percent of the noncarcinogenic or carcinogenic score in any medium, and therefore was retained for further consideration as a chemical of potential concern.

- Two potentially Site-related chemicals (viz., diphenyl sulfone and photomirex) had no USEPA-published toxicological values, and therefore are not excluded as chemicals of potential concern in this step.

- One potentially Site-related chemical, chloromethane, was detected only in two surface water samples at concentrations less than one half of the sample detection limit. At these levels, the estimated hypothetical cancer risk from consumption of 2 liters of water from the creek per day for 30 years is less

TABLE 7
Potentially Site-Related Chemicals Detected in Fish Ranked by Noncarcinogenic Score

Chemical	Risk Factor (Noncarcinogenic)
Mirex	3075000000
Di-n-Butylphthalate	14000
Acetone	8200
Methylene Chloride	5167
Butylbenzylphthalate	1800
Butanone (2-)	1140
Benzoic Acid	825
Tetrachloroethene	700
Phenol	633
Dimethylphthalate	210
Toluene	80
Ethylbenzene	50
Xylene (total)	10
TOTAL	3075032815

Note: Dashed line denotes scores greater than 1% of the total risk for this media.

TABLE 8
Potentially Site-Related Chemicals Detected in Sediment Ranked by Noncarcinogenic Score

Chemical	Risk Factor (Noncarcinogenic)
Mirex	840000000
Noncarcinogenic PAHs	145000
Methylphenol (4-)	56000
Acetone	800
Di(n)butylphthalate	740
Phenol	267
Butanone (2-)	200
Benzoic Acid	108
TOTAL	840203114

Note: Dashed line denotes scores greater than 1% of the total risk for this media.

TABLE 9
Potentially Site-Related Chemicals Detected in Fish Ranked by Carcinogenic Score

Chemical	Risk Factor (Carcinogenic)
Mirex	11070
Methylene Chloride	2
Tetrachloroethene	0.4
Benzene	0.1
TOTAL	11073

Note: Dashed line denotes scores greater than 1% of the total risk for this media.

TABLE 10
Potentially Site-Related Chemicals Detected in Sediment Ranked by Carcinogenic Score

Chemical	Risk Factor (Carcinogenic)
Mirex	3024
Dichloroethane (1,2-)	0.2
TOTAL	3024

Note: Dashed line denotes scores greater than 1% of the total risk for this media.

than one in one million [3]. [We note that any actual exposures and associated risks would be far smaller since the creek is not a source of drinking water, and the only anticipated potential exposures to creek water would result from occasional dermal contact and incidental ingestion when using the creek for recreational activities.] Because the concentrations detected in the MFLBC present minimal risks, chloromethane was eliminated as a chemical of potential concern.

C. Discussion of Chemicals Without Toxicity Data

After the previous step in the selection of chemicals of potential concern, two of the remaining three chemicals were without USEPA-published toxicity values. The two chemicals (viz., diphenyl sulfone and photomirex) are discussed individually below.

- **diphenyl sulfone.** The only toxicity information available for diphenyl sulfone is an acute intravenous LD_{50} of 320 mg/kg in the mouse (NIOSH 1990). This value is insufficient as a basis for deriving a chronic toxicity value for diphenyl sulfone. Therefore, because a quantitative assessment of risk cannot be conducted for this chemical given the inadequate toxicological data, diphenyl sulfone was eliminated as a chemical of potential concern.

- **photomirex.** A review of the toxicological data for photomirex is presented in Appendix B. For purposes of this assessment, the toxicity of photomirex is

---

[3] The cancer risk is estimated using the following equation:

R = C*I*ED*SF*

where

R = cancer risk
C = concentration (0.004 mg/l)
I = ingestion rate (2 l/day)
ED = exposure duration (30 years/75 years), where 30 years is considered an appropriate period of exposure and 75 years is an average lifetime (USEPA 1989a).
SF = slope factor (0.013 $(mg/kg/d)^{-1}$)
BW = body weight (70 kg)

considered to be equivalent to that of mirex. Photomirex is retained as a chemical of potential concern.

D. Consideration of Tentatively Identified Compounds (TICs)

Three TICs were detected in the surface water, thirty-five TICs were detected in the sediment, and seventy-four TICs were detected in fish. No TICs were detected in flood plain soil. None of the TICs observed in any media are known to have been produced or used at the Site. It is not possible to assess the risk presented by many of the TICs as they are not specifically identified (e.g., TIC compound C13H28). In addition, the estimated concentrations of many of the TICs are several orders of magnitude higher than any of the positively identified chemicals. USEPA guidance recommends that a TIC be eliminated from consideration if there is no historical information to suggest that a particular TIC may be present at the Site, or if the estimated concentration is very high and therefore is likely to dominate the estimated risks (USEPA 1989a). Based on this guidance, no TIC was retained as a chemical of potential concern.

E. Chemicals of Potential Concern

In summary, the two chemicals of potential concern identified for the MFLBC risk assessment are mirex and photomirex.

however RAGS p.5-18 also states that when many TICs are present relative to the TAL and TCL compds identified, or if TIC compds appear high or site info indicates TICs are indeed present, then further evaluation of TICs is necessary. Use SAS to confirm TICs.

Should do Exposure Assmt first so we can first determine all the exposure period for which toxicity values are necessary
Identify all exposure routes needed
(looks like inhalation toxicity totally overlooked here -- but we haven't even determined if its a valid exposure pathway

Dont know whether route to route extrapolations need to be ~~further~~ performed (know about oral → dermal)

Make sure all guidance consulted? - what about use of TCDD for mixes + photomixes

Dont know if sensitive subgroups were considered or need for other RfDs considered (ie. - developmental)

No discussion a summary of Toxicity
n description of Toxic adverse effects for mixes, photomixes, etc
no discussion of mutagenicity, dev., repro., + carcinogenic, etc.

IV. TOXICOLOGICAL ASSESSMENT

A. Introduction

In order to assess the potential health risk associated with exposure to chemicals in the MFLBC, it is necessary to examine the relevant toxicologic literature to determine the effects in humans or laboratory animals of chemical exposure as a function of exposure level. The USEPA has conducted such assessments on many frequently occurring environmental chemicals and has developed toxicity values for use in risk assessment based on these assessments. These toxicity values -- reference doses (RfDs) for noncarcinogenic chemicals and the noncarcinogenic effects of carcinogens, and cancer slope factors (SFs) for known, suspected, and possible human carcinogens -- are published by the Agency and updated regularly (USEPA 1990a, USEPA 1990b).

An RfD, generally expressed as a dose in mg/kg/day, is USEPA's estimate of the daily human exposure that is unlikely to result in deleterious effects following chronic exposure. Unless adequate human data are available, an RfD is generally based on a study from the most sensitive animal species tested and the most sensitive endpoint measured. From this critical study, the experimental exposure representing the highest dose level tested at which no adverse effects were demonstrated (the no-observed-adverse-effect level, NOAEL) is identified. The RfD is derived from the NOAEL for the critical toxic effect by dividing the NOAEL by uncertainty (or safety) and modifying factors. These factors generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the extrapolation from the available data. A 100-fold uncertainty factor is typically used when extrapolating results of long-term studies in experimental animals to humans, with additional factors applied where there are limitations in the available experimental data. The RfD derived by this process provides no sharp demarcation between "safe" and "unsafe" levels of exposure. In fact, exposures may even exceed the RfD without a significant risk arising. (See Chapter VIII, pp. 65-66 and 77-78 for additional discussion of the uncertainties in the development of the RfD.)

As noted above, the SF is the toxicity value developed for carcinogenic effects. An SF, expressed in units of $(mg/kg/day)^{-1}$, represents the upper 95% confidence limit on the linear component of the slope of the tumorigenic dose-response curve in the low-dose (low-risk) region. Unlike the RfD in noncancer risk assessment, the cancer SF is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a

lifetime and is derived by applying a mathematical model to extrapolate from the relatively high doses administered to experimental animals to the lower exposure levels expected for human contact in the environment. A number of low-dose extrapolation models have been developed. Each is based on general theories of carcinogenesis or certain statistical principles rather than on data for a specific chemical. USEPA generally uses the linearized multistage model in cancer risk assessment. This model has been used by USEPA to derive the SF for mirex. Other models are available, but generally are less conservative (i.e., predict lower cancer potency estimates) than the linearized multistage model. The latter model does not necessarily provide the most "correct" or "accurate" measure of carcinogenic potency, but is used by USEPA in part as a policy matter to provide a conservative (i.e., health protective) estimate of carcinogenic potency. (See Chapter VIII, pp. 78-80 for additional discussion of the uncertainties related to development of SFs.)

Reviews of the toxicity of mirex and photomirex and the toxicity values (i.e., RfDs and SFs) used in this assessment are presented in Appendix B. USEPA has developed an RfD and SF for mirex; these values and their bases are summarized in Tables 11 and 12. The USEPA toxicity values have been used in this assessment; however, as pointed out in Chapter VIII in the discussion of uncertainties associated with the risk assessment process (pp. 77-80), differences of opinion exist among scientists with respect to some of the underlying assumptions made in estimating these values. Furthermore, the risks estimated using these toxicity values must be interpreted in light of the conservative assumptions built into the toxicity values. These underlying assumptions are also discussed further in Chapter VIII, Risk Characterization.

USEPA has not derived toxicity values for photomirex. As discussed in Appendix B, the toxicity values for mirex have been applied to photomirex for purposes of this assessment.

B. Application of Oral SFs and RfDs to Assessment of Dermal Exposure

Because there may be differences in the absorption and pharmacokinetics of a chemical when exposure occurs by different routes, care must be taken to ensure that such differences are accounted for when RfDs or SFs derived from data on one route of exposure (in this case oral) are applied to another route (dermal). In the absence of detailed comparative absorption and pharmacokinetic data, a reasonable approach is to adjust either the toxicity measure (RfD or SF) or the exposure measure (estimated human intake) for the relative absorption via the route of interest compared to the route to which the toxicity measure applies.

TABLE 11
Summary of Chronic RfDs for Chemicals of Potential Concern (Oral Route)

Chemical	Chronic RfD (mg/kg-day)	Confidence Level	Critical Effect	RfD Basis/ RfD Source	Uncertainty and Modifying Factors
Mirex	2×10^{-6}	Low	Decreased pup survival in the vole	Diet/IRIS	UF = 10,000 (H, A, L, S, O) MF = 1

H = Variation in human sensitivity
A = Animal to human extrapolation
L = Extrapolation from LOAEL to NOAEL
S = Extrapolation of a subchronic effect to chronic equivalent
O = Other
IRIS = USEPA's online database: Integrated Risk Information System (USEPA 1990b).

TABLE 12
Summary of Carcinogenicity Slope Factors for Chemicals of Potential Concern (Oral Route)

Chemical	Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Weight of Evidence Classification	Type of Cancer	SF Basis/ SF Source
Mirex	1.8	B2	Liver, adrenal	Diet/HEAST

HEAST = Health Effects Assessment Summary Tables (USEPA 1990a).

B2 = Defined under USEPA's weight-of-evidence classification system for carcinogenicity as a "probable human carcinogen" based on "sufficient evidence in animals and inadequate or no evidence in humans" (USEPA 1989a).

are these oral values administered or absorbed doses? Assume Admin

There are two, mathematically equivalent, procedures for making this adjustment. One can adjust the toxicity measure (RfD or SF) to derive an **absorbed** RfD or SF, and use that value together with an estimate of **absorbed** dose by the route of interest. Thus, for example, if chemical X has an oral RfD of 2 mg/kg/day, and data indicate that 80% of an ingested dose of the chemical is absorbed, one can derive an absorbed RfD by multiplying the oral RfD by the fraction absorbed: 2 x 0.8 = 1.6 mg/kg/day. If one then wishes to examine the situation of dermal exposure to the chemical in soil, one can determine the dermal exposure (based on the quantity of soil contacting the skin and the concentration of the chemical in the soil), and then derive an **absorbed** dose for that pathway of exposure by multiplying the dermal exposure by the relevant dermal absorption factor. If, for example, the dermal exposure to chemical X is 0.5 mg/kg/day and the dermal absorption factor is 10% (0.1), the absorbed dose would be 0.5 mg/kg/day x 0.1 = 0.05 mg/kg/day. The hazard index (described in Chapter VIII, Risk Characterization) would then be calculated as:

$$\frac{\textbf{absorbed dermal dose}}{\textbf{absorbed RfD}} \quad \text{or} \quad \frac{0.05}{1.6} = 0.031.$$

Alternatively, rather than making one adjustment to the RfD and a second adjustment to the exposure, one can simply combine these two adjustments in a single adjustment factor applied to the exposure estimate to derive (in the case of the example used here) an equivalent oral dose and use the oral RfD without adjustment. This single adjustment corresponds to the **relative absorption** between the two exposure routes of interest. Using the example described above, the **relative** dermal absorption is calculated by dividing the **absolute** dermal absorption fraction (in this case 0.1, or 10%) by the **absolute** oral absorption fraction (in this case 0.8, or 80%): 0.1/0.8 = 0.125. This **relative** absorption factor is then multiplied by the dermal exposure estimate (in this case 0.5 mg/kg/day) to derive the **equivalent oral** dose from the dermal route: 0.5 x 0.125 = 0.063 mg/kg/day. The hazard index is then calculated as:

$$\frac{\textbf{oral-equivalent dermal dose}}{\text{oral RfD}} \quad \text{or} \quad \frac{0.063 \text{ mg/kg/day}}{2 \text{ mg/kg/day}} = 0.031.$$

Both of these procedures for making the adjustment for differences in absorption yield the same hazard index, 0.031 (i.e., they are mathematically equivalent). Similar procedures

apply when dealing with cancer slope factors. In this assessment we have used the latter procedure for making adjustments at the exposure end by incorporating data on relative absorption. We have chosen the relative absorption procedure for simplicity to avoid having to make two separate adjustments, and to avoid having multiple RfD values that might lead to confusion. Making the adjustment for differences in absorption is most critical in the case of dermal exposure, because dermal absorption is generally much lower than absorption via the oral or inhalation route.

Dermal absorption data for mirex and photomirex needed to derive a relative absorption factor for these chemicals are not available. In the absence of such data, this factor has been estimated based on absorption data for tetrachlorodibenzo(p)dioxin (TCDD), an environmentally persistent chlorinated organic compound with a high octanol-water partition coefficient (K_{ow}) similar to mirex and photomirex. Studies of dermal absorption of TCDD from soil indicate that dermal permeability is 1% or less (Poiger and Schlatter 1980; Shu et al. 1988; Driver et al. 1989). Ingested TCDD is largely absorbed, with gastrointestinal absorption reported to be 90% (Environment Canada 1984). Therefore, the relative absorption factor for mirex and photomirex, by analogy to TCDD, is estimated to be 0.01/0.9 or approximately 0.01.

.000002

2×10^{-6} mg/kg-day $\times$ 0.90 = 1.8×10^{-6} mg/kg-Day

V. IDENTIFICATION OF EXPOSURE PATHWAYS

A. Introduction

In this chapter, potential exposure pathways under current and hypothetical future conditions of the area along the MFLBC are identified. Exposure pathways are those situations by which a population or an individual could be exposed to chemicals present in the area along the MFLBC.

B. Characterization of Exposure Scenarios

Fundamental to the identification of exposure pathways is an understanding of the uses of the area along the MFLBC and the populations that may be exposed. For purposes of this baseline risk assessment, potential exposures under both current and hypothetical future uses of the area along the MFLBC are evaluated. A current exposure scenario evaluates whether there is a potential health threat under existing conditions. A future exposure scenario evaluates whether there is a potential health threat under hypothetical future conditions.

The identification of potentially exposed populations is based upon knowledge of existing chemical concentrations in the area along the MFLBC, local land-use patterns, activities of nearby residents, and judgments about what constitutes reasonable behavior. Populations potentially exposed to chemicals present along the MFLBC can be broadly identified as residents, recreational visitors, and farmers. Under both the current and future exposure scenarios, potential exposures to the following populations are characterized:

- Recreational visitors, who are assumed to engage in activities in and along the MFLBC; and

- Residents, whose property is located within the flood plain of the MFLBC.

Under the future exposure scenario, potential exposure is also characterized for:

- Farmers or an agricultural population, who are assumed to be raising livestock along the flood plain of the MFLBC. Potential exposures of farmers are not characterized under the current exposure scenario because Ruetgers-Nease installed fences and bridges on farms in 1989 to prevent access of livestock to

were these fences installed all along MFLBC? If not -- for current + future use

the MFLBC. Potential exposures to farmers are characterized under the future exposure scenario because USEPA guidance (1989a) requires that exposures be calculated as if these remedial measures were not put in place (i.e., in the absence of any actions to control or mitigate releases).

C. Identification of Potential Exposure Pathways

The identification of potential exposure pathways is primarily based upon site-specific information obtained from a survey conducted by ODH in September 1989. Data on potential exposure to mirex among persons living in the vicinity of the MFLBC were obtained through questions concerning fishing and recreational contact with the MFLBC, and consumption of game and farm products. On the basis of the survey, ODH concluded that the following were potential exposure pathways:

- Direct contact with the creek and its sediments during recreational activities such as fishing, swimming and wading. During these activities, potential exposure would be via incidental ingestion and dermal contact of sediments. As no chemical of potential concern was detected in surface water, ingestion [redacted]

- Ingestion of fish caught in the MFLBC by the recreational population. As above, exposures are likely to differ within and outside the advisory area.

- Ingestion of game hunted or trapped in the area along MFLBC by the recreational population. what types of game?

- Ingestion of vegetables grown in flood plain soil by residents. → what types?

- Ingestion of beef and milk from dairy cows with access to the MFLBC by farmers and their families. As discussed above, these exposure pathways are evaluated for the future exposure scenario only.

In addition to the potential exposure pathways identified by ODH, potential exposure could occur to the flood plain resident through dermal contact with and incidental ingestion of soils while gardening or playing.

Tables 13 and 14 summarize the pathways of exposure that may exist for the current and future uses of the area along the MFLBC.

TABLE 13
Routes of Potential Exposure Associated With the Middle Fork of Little Beaver Creek
Current Use

Exposure Medium/ Exposure Route	Flood Plain Resident	Agricultural Population[1]	Recreational Population
Soil			
Incidental ingestion	x		
Dermal contact	x		
Sediment			
Incidental ingestion			x[2]
Dermal contact			x[2]
Food			
Ingestion of vegetables	x		
Ingestion of beef			
Ingestion of milk			
Ingestion of fish			x[2]
Ingestion of game			x

[1] Flood plain on the property owned by two dairy farmers has been fenced. Under the current use scenario, therefore, cattle should have no access to these areas.

[2]There is currently an advisory against fishing and other water contact activities in the MFLBC above Lisbon Dam. Accordingly, exposure assumptions that apply to recreational activities and fishing along the stretch of creek above Lisbon Dam differ from assumptions that apply below Lisbon Dam under the current use scenario.

What about further downstream?

Tables should show
Receiving medium
Release mechanism
Release source
receptors

TABLE 14
Routes of Potential Exposure Associated With the Middle Fork of Little Beaver Creek
Future Use

Exposure Medium/ Exposure Route	Flood Plain Resident	Agricultural Population	Recreational Population
Soil			
Incidental ingestion	x		
Dermal contact	x		
Sediment			
Incidental ingestion			x
Dermal contact			x
Food			
Ingestion of vegetables	x	x	
Ingestion of beef		x	
Ingestion of milk			x
Ingestion of fish			x
Ingestion of game			

? What kinds of exposures are these? acute, chronic, adult, child?

No evaluation of chemical fate + transport after release!

VI. ESTIMATION OF ENVIRONMENTAL CONCENTRATIONS

A. Introduction

In order to assess the risk of exposure to chemicals at the MFLBC, an estimate of the concentration of chemicals of potential concern in the following environmental media is necessary:

- flood plain soil (surface - subsurface!)
- sediment
- fish
- vegetables
- beef and milk
- game
- Surface water!

Reasonable maximum exposure (RME) scenarios are evaluated as part of the baseline risk assessment. In accordance with USEPA guidance (1989a), the RME concentration is conservatively assumed to be represented by the 95 percent upper confidence limit on the arithmetic average (i.e., mean) concentration of the chemical of potential concern in the medium of interest. In other words, there is only a five percent probability that the true average is above the calculated RME concentration. This upper confidence limit was calculated using the methodology recommended by Gilbert (1987). In cases where this value exceeded the maximum detected concentration, the maximum detected concentration was used for the RME value.

For purposes of this assessment, the RME environmental concentrations for the flood plain soil, sediment, and fish media are based upon sampling data analyzed by Enseco-ERCO laboratories and validated by Environmental Standards Inc. The RME environmental concentrations for vegetables, beef, and milk are based upon models that relate these environmental concentrations to soil concentrations. These models have been used by USEPA regulators and scientists for assessments similar to the one conducted here. The RME environmental concentrations for game are based upon mirex concentrations found in wildlife along the MFLBC.

During the data validation process, Environmental Standards Inc. identified limitations in the analyses of several samples. In accordance with USEPA guidance (1989a), data were handled in the following manner:

- Unqualified chemical concentrations were used to calculate the RME concentration without modification.

- Where nondetect values were reported, chemicals were considered to be present at one half of the sample detection limit.

- Data marked with a R-qualifier, indicating an unreliable result, were not used to calculate the RME concentration.

- Data marked with a U-qualifier, indicating that the chemical was also detected in a blank, and data marked with a UL-qualifier, indicating that the chemical was not detected but the detection limit was probably higher due to a low bias identified during the quality assurance review, were treated the same as nondetects (i.e., at one half of the sample detection limit).

- Data marked with a J-qualifier, indicating that the concentrations were estimated, were treated the same as unqualified data.

B. Flood Plain Soil

The RME concentration for each of the chemicals of potential concern detected in flood plain soils are presented in Table 15.

C. Sediment

The RME concentrations for each of the chemicals of potential concern detected in sediment samples from the MFLBC are presented in Table 15. Sediment samples from background stations (viz., #1, #29, #30, #47, and #50) were not included in the calculation of the RME concentration. As discussed in the previous chapter, the sediment samples are grouped into those taken above and below the Lisbon Dam because the frequency of exposure in these areas is assumed to differ. how — why?

TABLE 15
Reasonable Maximum Exposure Concentrations
of Chemicals of Potential Concern in Fish, Sediment, and Flood Plain Soil

Chemical	Mean Concentration[1] (μg/kg)	Upper 95th% Confidence Limit of the Mean[2] (μg/kg)	Maximum Detected Concentration (μg/kg)	Reasonable Maximum Exposure Concentration (μg/kg)
Fish above the Dam				
Fillet				
Photomirex	8.9	12	28.8	12
Mirex	304.2	516	1820	516
Whole Body				
Photomirex	34.6	71	390	71
Mirex	870.1	1483	6150	1483
Fish below the Dam				
Fillet				
Photomirex	5.2	7	3.12	3.12
Mirex	24.2	33	67	33
Whole Body				
Photomirex	5.7	7	4.54	4.54
Mirex	33.0	44	65.5	44
Sediment above the Dam				
Photomirex	96	215	7.38	7.38
Mirex	214.2	320	1680	320
Sediment below the Dam				
Mirex	10.9	12	10.9	10.9

TABLE 15
Reasonable Maximum Exposure Concentrations
of Chemicals of Potential Concern in Fish, Sediment, and Flood Plain Soil

Chemical	Mean Concentration[1] (μg/kg)	Upper 95th% Confidence Limit of the Mean[2] (μg/kg)	Maximum Detected Concentration (μg/kg)	Reasonable Maximum Exposure Concentration (μg/kg)
Floodplain Soil				
Photomirex	17.8	25	132	25
Mirex	535.2	854	4540	854

[1]Because nondetect chemicals were considered to be present at one-half of the detection limit, mean concentration values are in some cases greater than maximum detected concentration values.

[2]Calculated according to methods described in Gilbert 1987, p. 139:

$$UL_{95} = \overline{x} + t_{95,n-1}s(n)^{-½}$$

where:

UL_{95} is the upper 95th % confidence limit of the mean
n is the sample size
$\overline{x}$ is the mean concentration
$t_{95,n-1}$ is the 95th % t distribution value for n-1 degrees of freedom
s is the standard deviation of the mean

D. Fish

The RME concentrations for each of the chemicals of potential concern detected in fish samples from the MFLBC are presented in Table 15. As with sediment, fish samples are grouped into samples taken above and below Lisbon Dam because the frequency of exposure in these areas is assumed to differ. In addition, fish samples are grouped into fillets and whole body samples because fillet samples would be representative of the fish concentrations available for human consumption.

E. Vegetables

Human exposure to chemicals present in the flood plain soils may occur through ingestion of vegetables grown in home gardens. To estimate the risks associated with consuming produce grown in home gardens, the concentration of chemicals of concern in vegetables are modeled. For this assessment, chemical uptake by three classes of vegetables was evaluated. These were:

- Leafy vegetables (e.g., cabbage and lettuce)
- Underground produce (e.g., carrots and potatoes)
- Non-leafy above ground produce (e.g., tomatoes, and cucumbers)

Because of the low volatility of mirex and the relatively small areal extent of soil contamination from which dust could be generated, the only pathway of plant uptake considered potentially significant is root uptake. Mirex uptake by plants through roots has been documented by de la Cruz and Rajanna (1975). The specific steps used to estimate root uptake of the two chemicals of potential concern are described below. The uptake equations used are based on published experimental data for mirex. No experimental data were available for determining crop uptake of photomirex. Given the similarities in structure between mirex and photomirex, the uptake factors developed for mirex are used to estimate crop concentrations of photomirex.

The chemical accumulation in plants from soil considers uptake from the soil through the root system and eventual movement to other plant parts. The equation for determining the dry weight concentration in the edible portion of the plants (C_p) is:

$$C_p = BC_s$$

where:

B = Soil/plant uptake factor

C_s = Concentration of the chemical in soil

For this assessment, uptake factors developed from the de la Cruz and Rajanna investigation are used to estimate the concentration of mirex in garden vegetables. The cited study determined mirex uptake by crops grown on two soil types, a sandy loam and a field soil of higher silt, clay, and organic matter content. Experimental results from the latter soil, which more closely represents flood plain soils along the MFLBC, are used for this assessment.

de la Cruz and Rajanna studied the uptake of mirex by four crops: garden beans, soybeans, sorghum and wheat. While only the beans are a typical garden vegetable, without additional data, these crops are assumed to be representative of those grown in home gardens. Uptake determined for the roots of these crops is assumed to be representative of underground produce from gardens (e.g., carrots and potatoes). Uptake determined for the leaves of these crops is assumed to be representative of normal leafy vegetables (e.g., cabbage and leaf lettuce). No uptake by non-leafy above ground produce (e.g., tomatoes) was presented in the de la Cruz and Rajanna study; as a default, the uptake factor developed for leaves is used for these vegetables.

Table 16 presents the mirex uptake factors for the roots and leaves of the four crop types grown in three concentrations of mirex in soils. It should be noted that the experimental soil concentrations ranged from 0.3 to 3.5 ppm, and encompass the RME flood plain soil concentration for mirex of 0.85 ppm. The average leaf and root uptake factors over all crop types and all mirex soil concentrations are combined to determine the final uptake factors for leaves and roots. These factors are 0.17 and 0.51 for leaves and roots, respectively.

The garden vegetable concentrations at the MFLBC are estimated by combining the soil/plant uptake factors with the RME flood plain soil concentrations along the MFLBC. As mentioned above, the modeled plant concentrations are in dry weight, and are converted to a wet weight basis assuming that the generic vegetable classes have the following fraction of water: underground produce, 0.828; leafy vegetables, 0.936; and non-leafy above ground produce, 0.879 (Baes et al. 1984). Table 17 presents the dry and wet weight vegetable concentrations resulting from the uptake calculations discussed above.

TABLE 16
Bioconcentration Factors for Mirex in Leaves and Roots of Four Plants at Various Soil Concentrations (de la Cruz and Rajanna 1975)

Plant Part	Soil Concentration (ppm)			
	3.5	0.8	0.3	Average of All Soils
Garden Beans				
Leaves	0.06	0.14	0.03	0.08
Roots	0.34	0.61	0.70	0.55
Soybeans				
Leaves	0.06	0.15	0.33	0.18
Roots	0.36	0.61	0.57	0.51
Sorghum				
Leaves	0.06	0.25	0.37	0.23
Roots	0.23	0.55	0.67	0.48
Wheat				
Leaves	0.05	0.22	0.30	0.19
Roots	0.33	0.34	0.77	0.48
Avg Leaf BCF over all crops 0.17 Avg Root BCF over all crops 0.51				

BCF is defined as the dry weight chemical concentration in the vegetable divided by the concentration in the soil.

TABLE 17
Wet and Dry Weight Garden Vegetable Concentrations Resulting from Root Uptake

Chemical of Concern	RME Soil Conc. (ppm)	Vegetable Concentration (mg/kg Dry Weight)			Vegetable Concentration (mg/kg Wet Weight)		
		Leafy Vegetables	Underground Produce	Above Ground Produce	Leafy Vegetables	Underground Produce	Above Ground Produce
Mirex	0.85	0.143	0.430	0.143	0.009	0.074	0.017
Photomirex	0.025	0.0042	0.0127	0.0042	0.0003	0.0022	0.0005

Dry weight concentrations are calculated using the bioconcentration factors for leaves (leafy vegetables and above ground produce) and roots (underground produce) presented in Table 16.

For dry weight to wet weight transformations, the average water contents for the generic vegetable classes were assumed to be 0.936 for leafy vegetables, 0.879 for non-leafy above ground produce, and 0.828 for underground produce.

As discussed above, root uptake is assumed to be the only major source of mirex and photomirex in crops grown in flood plain soils at the MFLBC. The vegetable concentrations to be used in assessing human exposure for this assessment are the wet weight concentrations presented in Table 17. For this assessment, the concentrations of the three vegetable types were combined to estimate a generic vegetable concentration using a weighted average based on the relative consumption rates of the vegetable types. For determining this weighted average, the relative consumptions of the vegetable types were assumed to be 9.1% underground produce, 86.2% non-leafy above ground produce, and 4.7% leafy vegetables (Pao et al. 1982). The weighted average vegetable concentration is presented in Table 18.

F. Beef and Milk

Human exposure to the chemicals of potential concern in flood plain soils can result indirectly from the consumption of locally raised cows. As previously mentioned, dairy cows are known to be raised on farms along the MFLBC. While beef cattle are not known to be raised in the area, for this assessment, consumption of beef from dairy cows is assumed to occur. To predict the risks associated with the consumption of beef and milk, the beef and milk concentrations of the two chemicals of concern found in flood plain soils, mirex and photomirex, are estimated. The uptake of chemicals in beef and milk are modeled using the methodology presented in Travis and Arms (1988). No experimental data was available for determining beef and milk uptake of photomirex. Given the similarities in structure between mirex and photomirex, the same uptake factors developed for mirex are used to estimate beef and milk concentrations of photomirex.

Biotransfer factors for beef (B_b) and milk (B_m) were used to estimate chemical concentrations in these foods. They are defined as follows (Travis and Arms 1988):

Uncertainty

B_b = Concentration in beef (mg/kg)/daily intake of compound (mg/d)

B_m = Concentration in milk (mg/kg)/daily intake of compound (mg/d)

The B_b and B_m values for mirex (0.056 and 0.0095, respectively) are the experimentally determined values presented in Travis and Arms. These values were derived from experimental studies of mirex conducted by Bond and Woodham (1975) and Dorough and Ivie (1974) and have been adopted for purposes of this assessment.

Ingestion of feed and inadvertent ingestion of soil while grazing on flood plain fields are assumed to be the major sources of chemical intake. The dietary assumptions used for

TABLE 18
Weighted Average Vegetable Concentrations

Chemical of Concern	Vegetable Concentration (mg/kg Wet Weight)			
	Leafy Vegetables	Underground Produce	Non-Leafy Above Ground Produce	Weighed Average Vegetable[1]
Mirex	0.009	0.074	0.017	0.022
Photomirex	0.0003	0.0022	0.0005	0.0007

[1] The weighted average vegetable concentration was determined using the following relative consumptions; leafy vegetables 4.7%; non-leafy above ground produce 86.2%; and underground produce 9.1%.

this assessment (Travis and Arms 1988, Fries et al. 1982) are shown below:

Ingestion rate of dry feed:	16 kg/day
Ingestion rate of soil:	0.7 kg/day

As only a portion of the farmland along the MFLBC is located in the flood plain, 25% of the feed and soil ingested by the dairy cow is assumed to contain mirex and photomirex. This is considered to be a conservative estimate since farmers generally only put their cattle out to pasture for limited periods during the year (e.g., May through November) and since each farm has different patterns for which cattle might have access to MFLBC.

The concentrations of mirex and photomirex in dry feed are assumed to be the same as that modeled in Section E for dry weight leafy vegetables (0.143 ppm and 0.0042 ppm for mirex and photomirex, respectively). The concentrations of mirex and photomirex in soils are assumed to be the RME flood plain soil concentration (0.85 ppm and 0.025 ppm for mirex and photomirex, respectively). Multiplying these concentrations by the above intake rates and the fractions of feed and soil from the flood plain results in daily intake rates for the two chemicals of concern of:

Daily intake of mirex	0.72 mg/day
Daily intake of photomirex	0.021 mg/day

Applying the bioconcentration factors for beef and milk, the concentrations of mirex and photomirex in fresh meat and whole milk are estimated to be:

	Fresh Meat (ppm)	Whole Milk (ppm)
Mirex	0.040	0.0068
Photomirex	0.0012	0.00020

G. Game

Residue data for mirex in wildlife samples collected by the ODH were used for estimating concentrations of mirex in game. The ODH took samples of blood and fat from raccoon and opossums at nine sites along the MFLBC in September and October 1989.

Trapping stations were located along the length of the creek from near the Site down to Beaver Creek State Park. Raccoon and opossum were chosen for this survey because the diet of these animals, which consist of a wide variety of plants and animals including fish, crayfish, and other aquatic animals, would provide the greatest potential for bioaccumulation of mirex.

The results of the ODH wildlife sampling are included in Appendix A. Mirex levels in fat samples from the 22 animals trapped near the MFLBC ranged from non-detect (in 8 samples) to 0.0527 ppm; levels in blood (serum) samples ranged from non-detect to 0.0089 ppm. The detection limit for the mirex analyses was not provided. In the absence of a reported detection limit, the average concentration in fat of 0.0153 ppm was calculated by averaging only the samples with detectable mirex concentrations.

Because mirex partitions into fat, the concentration in the edible portion (i.e., meat) of game was estimated from residue data in fat based on the following: the percent fat in raccoon meat (cooked, roasted) is 14.5% (USDA 1975) and the percent fat in separable fat (estimated from data for various cuts of beef) is about 75 percent (USDA 1975). Accordingly, the average mirex level in raccoon fat of 0.0153 ppm can be adjusted by the ratio 0.145/0.75 to give an estimated concentration in the edible portion of game of 0.003 ppm.

Other game such as deer would likely have substantially lower mirex residue levels because of differences in the components of the diet and lower fat content in the edible portion (i.e., the fat content of deer meat is reported to be 4 percent, USDA 1975). Therefore, the concentration derived for game of 0.003 ppm is considered highly conservative for most hunters. It should be noted that the ODH reviewed the wildlife residue data and concluded the following: "We do not believe that consumption of raccoons and opossums hunted or trapped in the MFLBC watershed poses a significant risk to human health". The ODH has also pointed out that while no federal standards exist for mirex levels in wildlife, the range of detectable levels is considerably lower than the federal recommendation for commercial meat of 100 ppb (or 0.1 ppm). (See Appendix A.)

VII. ESTIMATION OF HUMAN INTAKE

A. Introduction

The next step in the risk assessment process is the estimation of the human intake received through exposure to the chemicals of potential concern in the various environmental media. Chemical intakes (also referred to as Chronic Daily Intakes or CDIs) are expressed in terms of the mass of substance in contact with the body per unit body weight per time (or mg/kg/day), and are calculated as a function of chemical concentration in the medium, contact rate, exposure frequency and duration, body weight, and averaging time. The values for some of these variables are dependent upon conditions specific to the site and characteristics of the potentially exposed populations.

It is not possible to estimate accurately the exposures for potentially exposed populations due to uncertainties in both current and future behavior patterns of these populations and limitations in knowledge of other exposure variable values. In light of this uncertainty, USEPA (1989a) recommends that intakes reflect an estimate of the reasonable maximum exposure (RME), defined as the highest exposure that is reasonably expected to occur. USEPA's intent with the RME is to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposures. As discussed in the Exposure Factors Handbook (USEPA 1990c), USEPA recommends that not all values be at their individual maximum in calculating the RME; professional judgment can be used to combine values to arrive at a set of variables that adequately estimates the RME. Consistent with USEPA guidance (USEPA 1989a, 1990c), the estimates of human intake calculated in this risk assessment are those for an RME.

Default exposure factors

In an exposure assessment, it is generally necessary to provide at least two different estimates of the CDI, one for noncarcinogenic effects and a second for carcinogens. The CDI generally used in the assessment of noncarcinogenic effects is the average daily dose an individual is likely to receive on any day during the period of exposure. In cases where exposure is intermittent, USEPA guidance states that it is appropriate to average the intake over the period of exposure. For carcinogens, the CDI is estimated by averaging the total

Because of the uncertainty assoc. with any estimate of exposure concentration, the UCL (95%) on the arithmetic average will be used for variable - assume log-normal distrib.

cumulative intake over a lifetime (USEPA 1989a).[4] This distinction in the calculation of the CDI for carcinogens and noncarcinogens relates to the currently-held scientific opinion that the mechanism of action of the two categories of chemicals is different. For carcinogens, the assumption is made that a high dose received over a short period of time produces a carcinogenic effect comparable to a corresponding low dose spread over a lifetime (USEPA 1989a).

As previously described, the exposure assessment considers the following potentially exposed populations and exposure scenarios:

- Flood plain residential population--current and future use
  - Potential exposure to soil and homegrown vegetables
- Recreational population--current and future use
  - Potential exposure to MFLBC sediment, fish, and game
- Farmers or an agricultural population--future use only
  - Potential exposure to homegrown beef and milk

Among the exposure assumptions used in the estimation of intake for these three populations is the assumption that exposures occur for 30 years. An exposure duration of 30 years is estimated by USEPA (1990c) to approximate the 90th percentile value for length of time a homeowner will live at one residence. USEPA (1989a) recommends that this value be used in estimating the reasonable maximum exposure. It should be noted that the majority of the U.S. population (i.e., approximately 90 percent) lives at one residence for less than 30 years and that the average length of residence at one home is approximately nine years.[5]

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[4]Averaging time (AT) for noncarcinogens and carcinogens will differ as follows: For noncarcinogens, the AT is the period over which exposure is assumed to occur (i.e., ED x 365 days/year). For carcinogens, intakes are calculated by prorating the total cumulative dose over a lifetime (75 years). Therefore, the AT equals 75 years x 365 days/year or 27375 days.

[5] Estimates of cancer risk are directly proportional to the duration of exposure. The duration of exposure used in this baseline risk assessment of 30 years is well above the average length of residence at one home of 9 years. Cancer risk estimates based on an average length of residence of 9 years would be less than one-third those estimated in this assessment.

In this assessment, exposures were modeled for the following four age groups: ages 1 to 6 (represented by a 4-year old), ages 6 to 12 (represented by a 9-year old), ages 12 to 18 (represented by a 15-year old), and ages 18 and above (i.e., adult). It was necessary to model exposures for specific age groups because the contact rates for several of the exposure pathways, notably incidental soil ingestion and consumption of milk, are substantially higher for children and teenagers, resulting in higher estimated intakes (in mg/kg/day) for these groups than for adults.[6]

Where appropriate, specific exposure assumptions have been adopted from USEPA guidance (1989a, 1990c). Where USEPA guidance was either incomplete, not specific to the age groups modeled, or not relevant to this assessment, additional sources of exposure information (e.g., Pao et al. 1982) were used. One valuable source of site-specific information on frequency of exposure was obtained from a survey conducted by the ODH.

In September 1989, the ODH conducted a survey of area residents to determine the extent of exposure to mirex among persons living in the vicinity of the Site and MFLBC. Subjects, chosen based on their proximity to the creek, were obtained by mailing questionnaires to 575 area families and placing 100 questionnaires in area libraries, as well as through announcements in three local newspapers and at public meetings. Data on potential exposure were obtained through questions concerning fishing and recreational contact with the creek, consumption of game and farm products, and employment history. The survey and results of the survey provided to ENVIRON by the ODH are included in Appendix C.

Because this survey provides site-specific information on the extent of potential exposure of local residents to MFLBC, these data were used, as appropriate, in preference to default assumptions recommended in USEPA guidance. The specific exposure variable values derived from the ODH survey data are described in Appendix D. In using these data, however, consideration was given to certain limitations of the survey for use in the context of a risk assessment. Because the study was conducted specifically to identify those members of the local population believed most likely to have been exposed to mirex, the response from study participants may not be representative of all area residents. Of 675 distributed questionnaires, only 200 families responded, resulting in a response rate of 30%. One further limitation of the study is that the exposure information provided by survey

---

[6]Because age-specific exposures were modeled, the exposure duration (ED) of 30 years was divided among the four age groups as follows: ages 1-6, ED=5 years; ages 6-12, ED=6 years; ages 12-18, ED= 6 years; and adult, ED=13 years.

respondents, who were most likely adult heads-of-household, may not have represented in all cases those exposures of all family members. Despite the limitations in the ODH survey for purposes of characterizing exposure variables in this assessment, the survey data were considered to provide a reasonable measure of exposure frequency and have been taken into account in the assessment of exposure to fish, game, and MFLBC sediments.

The remainder of this section presents the equations and assumptions used to calculate human intake associated with each relevant exposure pathway. An explanation of the basis for the exposure variables values used in the equations is presented in Appendix D.

B. Ingestion of Soil and Sediment

Exposure to chemicals of potential concern is assumed to occur through incidental ingestion of soil by the current and future flood plain resident (both adults and children) and through ingestion of sediments by the current and future recreational population. The following factors must be considered when estimating exposure via this pathway: 1) the chemical concentration in soil or sediment; 2) the rate of soil or sediment ingestion; 3) the period of time over which soil or sediment ingestion occurs; and 4) the bioavailability of the chemical adsorbed to soil or sediment, if known.

Exposure of flood plain residents to known or suspected carcinogenic and noncarcinogenic chemicals of potential concern via soil ingestion were calculated using the equation and assumptions in Table 19.

As a result of the advisory posted along the MFLBC, the frequency of visits to the MFLBC in the area of the advisory and downstream of the advisory (i.e., upstream and downstream of Libson Dam) is likely to differ. The frequency of visits within the advisory area may also differ in the future. Current exposure to known or suspected carcinogenic and noncarcinogenic chemicals of potential concern via ingestion of sediment upstream of Libson Dam (i.e., in the area of the advisory) were calculated using the equation and assumptions in Table 20. Potential exposures resulting from ingestion of sediment currently downstream and in the future in both upstream and downstream sections of the creek were calculated using the assumptions in Table 21.

APPENDIX A
Ohio Department of Health Wildlife Sample Results

246 N. High Street
Post Office Box 118
Columbus, Ohio 43266-0118

Telephone 614/466-0343

RICHARD F. CELESTE
Governor

November 26, 1990

Steven W.Foard
Ruetgers-Nease Chemical

201 Struble Rd.
State College, PA 16801

OHIO DEPARTMENT OF HEALTH WILDLIFE SAMPLE RESULTS

Fish samples taken in 1984 and 1987 in the Middle Fork of Little Beaver Creek (MFLBC) indicated that fish in the creek contained substantial amounts of mirex from the Ruetgers-Nease Chemical Company Superfund Site in Salem, Ohio. The Ohio Department of Health issued a fish consumption advisory in 1987 and a contact advisory in 1988. This evidence raised concerns about the presence of mirex in other animals in the area of the site. In September and October 1989, the Bureau of Epidemiology and Toxicology took samples of blood and fat from raccoons and opossums at nine sites along the Middle Fork of Little Beaver Creek. These samples were taken to determine if animals other than fish had picked up mirex from their environment. The wildlife sample sites were located along the length of the creek, from near the superfund site downstream to Beaver Creek State Park.

Raccoons and opossums were chosen because these animals are the most likely to accumulate mirex in their bodies. Raccoons and opossums typically live and forage for food around creeks, rivers, lakes and wetlands. They eat a wide variety of animals and plants, including fish, crayfish, and other aquatic animals.

In areas treated with mirex to control fire ants (primarily in southern United States) residues in mammals were highest in carnivores (meat eaters, such as foxes) and insectivores (eats insects), lower in omnivores (such as raccoons and opossums) and lowest in herbivores (eats plants, such as rabbits). In other studies where mirex was applied for fire ant control, mirex concentrations were highest in omnivores and carnivores in both aquatic and terrestrial ecosystems. Most mammals living in areas treated with mirex contained mirex residues. Concentration tended to reach a maximum soon after application and declined significantly during the 12 months following.

Blood and fat samples from raccoons and opossums trapped near MFLBC contained mirex ranging from essentially none to 52.7 parts per billion (ppb) in an opossum fat sample (Table 1). Most of the samples had very low levels of mirex. The average mirex concentration in blood was 2.19 ppb and in fat, 9.17 ppb. The highest levels were in fat samples (52.7 ppb and 39.9 ppb) of animals taken closest to the site. Mirex concentrations were generally lower in animals further downstream. The variations in concentration may have been a result of animal size or age. Larger and older animals would be expected to have higher concentrations of mirex. There were also a couple of raccoon and opossum samples taken at downstream sites with slightly higher concentrations. Fish samples in this general area of the creek also contained increased levels of mirex and may have contributed to increased levels in the wildlife samples.

In published studies analyzing wildlife from areas treated with the pesticide, mirex concentrations were generally higher than what was found in samples taken along MFLBC. Mirex concentrations in some of these studies were 1000 times greater than in our samples (Table 2).

There are no federal or state regulations for allowable concentrations of mirex in sport hunted or trapped (noncommercial) wild game, however, the Federal Food and Drug Administration tolerance level for mirex in commercial meat is 100 ppb. Mirex levels in ODH's study did not approach this level. If, however, the consumer is concerned they may choose to hunt or trap another type of animal or trim the fat from these animals. Mirex concentrations would be highest in the fat. We do not believe that consumption of raccoons and opossums hunted or trapped in the MFLBC watershed poses a significant risk to human health. Mirex concentrations in raccoons and opossums in Ohio were very low compared to animals in areas of the southern U.S.

Respectfully,

Tracy Shelley

Tracy L. Shelley, M.S.
Chief
Health Assessment Branch
Bureau of Epidemiology
& Toxicology
Ohio Department of Health
P.O. Box 118
Columbus, OH 43266-0118
(614) 644-6447

Kim Mortensen

B. Kim Mortensen, Ph.D.
Chief
Bureau of Epidemiology
& Toxicology
Ohio Department of Health
P.O. Box 118
Columbus, OH 43266-0118
(614) 466-5599

TLS:BKM/jsa/WILD.LET

TABLE 1

ODH WILDLIFE SAMPLES

Sample Site	Sample Type	Mirex Concentration (ppb)
1	A Raccoon Fat	39.9
	Serum	2.5
	B Opossum Fat	52.7
	C Opossum Fat	ND
2	A Raccoon Serum	4.5
	Fat	6.5
	B Raccoon Serum	0.7
	Fat	1.7
3	A Raccoon Serum	0.4
4	A Raccoon Serum	1.1
	Fat	ND
	B Opossum Serum	0.6
	Fat	9.6
5	A Raccoon Serum	5.9
	Fat	ND
	B Opossum Serum	6.4
	C Opossum Serum	ND
	Fat	23.7
	D Opossum Serum	8.9
6	A Opossum Serum	2.6
	Fat	ND
	B Raccoon Fat	4.3
7	A Raccoon Fat	ND
	B Opossum Fat	4.9
	C Opossum Serum	3.3
	Fat	9.5
	D Opossum Serum	7.5
	Fat	13.7
8	A Raccoon Fat	1.4
	B Raccoon Serum	5.4
	Fat	ND
	C Raccoon Fat (Road Kill)	ND
9	No Samples	
10	Raccoon Serum	0.6
	Fat	ND

Table 2
Mirex Concentration in Wildlife
from the
Southern United States

Animal	Mirex Concentration (ppm)*
Coyote fat	6.0 ppm
Opossum fat	9.5 ppm
Raccoon fat	75.9 ppm
Shrews	41.3 ppm
Frogs	9.0 ppm
Lizards	5.5 ppm
ODH-Raccoon fat	52.7 ppb**

*Parts Per Million

**Parts Per Billion

246 N. HIGH STREET
POST OFFICE BOX 118
COLUMBUS, OHIO 43216-0118

NEWS RELEASE

RICHARD F. CELESTE
Governor

RONALD L. FLETCHER, M.D.
Director of Health

November 26, 1990

FOR IMMEDIATE RELEASE

MINIMAL RISK SEEN FROM BANNED PESTICIDE FOUND IN WILDLIFE

COLUMBUS - Varying degrees of contamination from a banned pesticide found in raccoons and opossums along the Middle Fork of Little Beaver Creek in Mahoning and Columbiana counties does not pose a significant risk to human health, according to an Ohio Department of Health study released today.

The study included nine sites along the creek watershed from the former Nease Chemical Company site near Salem to Beaver Creek State Park. Results showed varying levels of mirex in the blood and fat of the animals.

The average mirex concentration found in blood was 2.19 parts per billion while the average level found in fat was 9.17 parts per billion. The highest levels were found in fat (52.7 ppb and 39.6 ppb) at sites closest to the former chemical company site. Concentrations tended to be much lower in downstream samples, although no pattern of decrease was noted.

While no federal standards exist for mirex levels in wildlife, the range of detectable levels of mirex found in those animals studied ranged from not being detectable to 52.7 parts per billion. These readings were considerably lower than the federal recommendation for commercial meat (100 parts per billion).

Compared to areas of the southern United States where mirex was widely used, the levels of mirex found in this wildlife study were very low.

Eating wild game is considered an individual choice and not regulated by any state or federal agency. While the Ohio Department of Health does not feel eating raccoons or opossums hunted or trapped from the creek watershed to be a significant risk, concerned consumers may want to hunt or trap animals other than raccoons or opossums or to trim fat from animals prior to eating.

Mirex has previously been found in fish samples taken from the creek and in sediment, prompting the department to issue a fish consumption advisory during 1987 and a contact advisory in 1988.

A December 1989 study of 42 people likely to be exposed to mirex showed those who had detectable levels of the pesticide in their blood either worked at the former chemical company or ate contaminated farm animal products.

A pesticide used to control imported fire ants, mirex was among pesticides produced at the site from 1961 until the Ohio Environmental Protection Agency forced the company to close i 1973. The site was placed on the United States Environmental Protection Agency National Priority List (Superfund) in 1983.

Because there is no data on the effects of exposure to mirex from the environment or throug eating fish or animal products, the Ohio Department of Health recommends people to limit or reduce their exposure.

-30-

Contact: Richard L. Wittenberg, (614) 644-8562

THE VINDICATOR - Youngstown
Friday, November 30, 1990
Pages B1 and B2

Official: Trapped animals should be safe to consume

■ Tests have found traces of mirex in animals trapped along the Middle Fork of Little Beaver Creek, but state health officials say the animals should be safe to eat.

By BOB JACKSON
VINDICATOR SALEM BUREAU

SALEM — Mirex, a suspected cancer-causing chemical, has apparently made its way into the food chain along the Middle Fork of Little Beaver Creek, an Ohio Department of Health report said.

However, the ODH says the levels of mirex found in raccoons and opossums trapped along the creek do not pose a significant health risk to humans who eat the animals.

Tracy Shelley, environmental scientist for ODH, said mirex levels in the animals ranged from non-detectable to 52.7 parts per billion. The tests checked for mirex in the blood and fat of the animals.

Averages: The average mirex concentration in blood was 2.19 ppb, and the average mirex content in fat was 9.17 ppb, Ms. Shelley said. Those numbers are well below the tolerance level of 100 ppb for commercial meats, which is what lead the ODH to issue its finding that the mirex poses no significant health risk.

"It's not necessarily a good comparison, but the commercial tolerance levels are the only thing we have to compare with. No federal standard exists," she said.

Ms. Shelley said the ODH recommends that people eat animals caught along the creek at their own risk. She said 11 raccoons and 10 opossums were taken from nine different locations along the creek. They were tested more than a year ago.

Randy Hertzer, a spokesman for the ODH, said if people are concerned, they should either hunt wild game in areas other than

Please see ANIMALS page B2

■ ANIMALS/*Safe to eat*

CONTINUED FROM PAGE B1

along the creek, or be sure to trim the fat off of any animals caught near the creek. He said mirex showed a greater tendency to settle in the fat.

Hertzer said there may be some meat contamination, but said it should be much lower than what is in the fat and blood.

Both Hertzer and Ms. Shelley said there is not enough evidence to indicate what long-term effects there are from exposure to or ingesting mirex.

Mirex is a suspected carcinogen that was manufactured at the former Nease Chemical plant on Benton Road, north of Salem. It has been detected in fish and sediment from the creek, and in some cattle that grazed near the creek.

The ODH issued a fish consumption advisory in 1987, and, in 1988, an advisory against coming into contact with the creek.

APPENDIX B
Toxicological Profiles

MIREX

A. Summary

Information on the toxicity of mirex in humans is limited. Although cases of human exposure are known, there are no reports of adverse health effects in humans associated with mirex exposure.

Experimental animal studies have shown that mirex is partly absorbed from the gastrointestinal tract (estimates of absorption range up to 85%) and is excreted primarily in the feces. Urinary elimination accounts for less than 1% of the total dose. Once absorbed, it is readily distributed throughout the body, with accumulation predominantly in fat. There is no evidence to suggest that mirex is metabolized once it is absorbed into the body. The excretion of mirex is slow and can last up to 100 days. The acute oral LD_{50} of mirex in rats is reported to be 740 mg/kg in males and 600 mg/kg in females. The toxic effects of mirex in subchronic rodent studies are characterized by decreased body weight and increased liver weight accompanied by morphological changes in the liver cells. Mirex induces microsomal liver enzymes and produces a proliferation of smooth endoplasmic reticulum. Mirex has been shown to be carcinogenic to both rats and mice. In a two-year NTP (1990) feeding study, there was clear evidence of carcinogenicity for male and female F344/N rats, as indicated by increased incidences of benign neoplastic nodules of the liver, as well as by increased incidences of pheochromocytomas of the adrenal gland and transitional cell papillomas of the kidney in males and by increased incidences of mononuclear cell leukemia in females. Various reproductive effects of mirex administration in animals have been reported, including reductions in fertility and litter size, and decreased birth weight and survival of offspring. Mirex has been found to cause various fetal abnormalities in laboratory animals. Mirex was negative for mutagenic potential in a variety of tests with both prokaryotic and eukaryotic cells.

The RfD for chronic oral exposure to mirex is reported by USEPA to be 2×10^{-6} mg/kg/day. The slope factor for carcinogenicity is 1.8 $(mg/kg/day)^{-1}$. Under USEPA's weight-of-evidence classification system for carcinogenicity, the Agency has characterized the available carcinogenicity data for mirex as "inadequate or no evidence in humans" and "sufficient evidence in animals". This characterization corresponds to a B2 carcinogen, "probable human carcinogen" under the USEPA classification scheme (USEPA 1989, 1990).

B. Health Effects in Humans

The extensive use of mirex for control of the fire ant has resulted in documented cases of environmental exposure. Kutz et al. (1974) reported lipid levels of mirex of 0.16 to 5.94 ppm (mean = 2.49 ppm) in 6 samples from 1400 samples collected as part of the National Human Monitoring Program for Pesticides (April 1971 to April 1972). Mirex was detected at a frequency of 10.2% in a study of persons living in the southeastern U.S. where large amounts of the pesticide had been applied. The geometric mean lipid concentration of mirex in these samples was 286 ppb (Kutz et al. 1985). Bush et al. (1983) found mirex present at very low concentrations (0.1 ng/g wet weight whole milk) in human milk samples from women living in upstate New York. In another study, mirex was found in 3 out of 14 samples of human milk (Mes et al. 1978). Burse et al. (1989) used concentrations of mirex in serum and adipose tissue from human subjects to calculate paired adipose tissue and serum ratios. The mean concentration ratio for mirex in adipose tissue to mirex in serum (whole weight basis) was 364 with an SE of 57 (95% CI = 252 to 476). Blood samples collected from pregnant women in the Jackson and Mississippi Delta areas were analyzed for several pesticides. The number of samples positive for mirex was 106/142 with a mean of 0.54 ppb (Lloyd et al. 1974). In a study by Murphy et al. (1983), the occurrence of selected pesticide residues in human blood serum from 4200 specimens of persons in 54 locations gave a frequency of detection for mirex of $<1\%$. There is no information concerning mirex levels in persons with occupational exposure.

Despite known exposure, there are no reports of adverse health effects associated with exposure to mirex in humans. The only epidemiological study available concerning mirex was of residents from an area in upstate New York that had been contaminated with several pesticides. There was no evidence of higher cancer incidence associated with residence in this area (Janerich et al. 1981).

C. Health Effects in Animals

1. Pharmacokinetics

Studies in laboratory animals demonstrate that mirex is partly absorbed from the gastrointestinal tract after an oral dose, with the remainder of unchanged mirex excreted primarily in the feces. Mehendale et al. (1972) reported that approximately 55% of a single oral dose of 6 mg ^{14}C-mirex/kg body weight administered to rats was excreted in the feces within 48 hours. Gibson et al.

(1972) reported that only 15% of a 0.2 mg/kg dose of mirex given to rats was excreted in the feces within 48 hours. This corresponds to an absorption of 85% of the administered dose. A single oral dose of 38 μg mirex to rats resulted in fecal elimination of 25-31% of the total dose within 2 weeks which represents over 70% absorption (Chambers et al. 1982). Byrd et al. (1982), using a pharmacokinetic model for rats, calculated an average absorption for mirex of 69% from a 1 mg/kg oral dose. In a study using a female rhesus monkey, a single oral dose of 1 mg/kg ^{14}C-mirex was detected in the plasma after 2 hours and reached a peak after 5 hours (Wiener et al. 1976).

Once absorbed, mirex is readily stored in the body. Mehendale et al. (1972) reported that after a single oral dose to rats of 6 mg mirex/kg, 34% of the total dose was retained, of which 28% was in fat, 3.2% in muscle, 0.09% in the kidneys, and 1.8% in the liver. Rats and Japanese quail fed diets containing ^{14}C-mirex (0.3, 3, 30 mg/kg) for 16 months accumulated levels of mirex in the fat at 120 to 185-fold greater than the dietary intake, with no plateau observed in the accumulation. An additional 10 months on a normal diet resulted in only a 40% decline in tissue concentration. Mirex has been shown to cross the placental barrier and accumulate in the rat fetus (Gaines and Kimbrough 1970).

Mirex does not appear to be metabolized in any animal species investigated (mice, rats, rabbits, monkeys) (WHO 1984).

Mirex is excreted primarily in the feces of experimental animals. There is very little excretion in urine; 0.7% of a 6 mg/kg dose was excreted in urine within 48 hours (Mehendale et al. 1972). Chambers et al. (1982) reported <1% urinary excretion of a 38 μg dose of mirex within 2 weeks. Excretion kinetics appear to be biphasic with a fast initial phase of 38 hours and a slow phase projected to last up to 100 days (Mehendale et al. 1972). Pittman et al. (1976) predicted a long half-life for mirex in rhesus monkeys; using a mathematical model, a 2% decline in adipose tissue levels of mirex would occur over a ten year period.

2. Acute Effects

The acute oral LD_{50} (i.e., the dose calculated to cause mortality in 50 percent of the test animals) for mirex in rats is reported to be 740 mg/kg for males and 600 mg/kg for females (Gaines 1969). The acute oral LD_{50} in male mongrel dogs was >1000 mg/kg (Larson et al. 1979).

3. Subchronic Effects

The effects of short-term administration of mirex in animals are characterized by decreased body weight (Kendall 1974; Villeneuve et al. 1977), increased liver weight (Villeneuve et al. 1977; Mehendale et al. 1973; Robinson and Yarbrough, 1968; Kaminsky et al. 1978; Abston and Yarbrough 1976; Davison et al. 1976), morphological changes in liver cells such as hepatocyte enlargement, depletion of glycogen and lipid accumulation and cell necrosis (Kendall 1974, 1979; Davison et al. 1976, Villeneuve et al. 1977) and induction of mixed-function oxidases (Villeneuve et al. 1977; Kaminsky et al. 1978; Iverson 1976; Warren et al. 1978; Baker et al. 1972; Davison and Cox 1974; Fouse and Hodgson 1987; Crouch and Ebel 1987). Increased liver weight in rats is accompanied by increased incorporation of [^{3}H]thymidine into DNA, at 36 or 48 hours after exposure to mirex (Yarbrough et al. 1986). The liver enlargement is dependent on an intact pituitary-adrenalcortical axis and requires corticosterone (Ervin and Yarbrough 1985). Impairment of hepatobiliary function (Mehendale 1976, 1977, 1979; Mehendale et al. 1979) and bile stasis (Gaines and Kimbrough 1970) have also been noted in laboratory animals.

Gaines and Kimbrough (1970) conducted a study using Sherman rats (10/sex/group) given mirex in the diet at 0, 1, 5, or 25 ppm for 166 days. Effects were noted in the liver, with increased relative liver weight at 25 ppm and slight hepatocellular hypertrophy in males at 1 ppm and females at 5 ppm.

A study by Larson et al. (1979) investigated the toxicity to Charles River rats of mirex given in the diet for 13 weeks at levels of 0, 5, 20, 80, 320, or 1280 ppm (10 rats/sex/group). In addition, beagle dogs (2/sex/group) were administered a diet containing 0, 4, 20, or 100 ppm mirex for 13 weeks. In the rat study, all females and five males in the 1280 ppm group died. There was one death in the 320 ppm group (female). Growth was depressed in both sexes in the 1280 ppm group and in males in the 320 ppm group. Hematologic effects were not apparent in any treatment group below 320 ppm at 13 weeks; however, at 13 weeks, hemoglobin values were depressed at 320 and 1280 ppm in both sexes and total white cell counts were elevated in the males from the 1280 ppm group. Significant hepatomegaly was present in males from the groups given 80 ppm and above and in females given 320 ppm. (Data were not obtained from females in the 1280 ppm group, all of which died during the course of the study.) Histopathology revealed liver cell changes in rats receiving 80 ppm and above.

Abnormalities were not observed in other tissues. In the dog study, two animals from the high dose group died during the study (one male and one female). Weight gains were comparable to control in the low and mid dose and decreased at the high dose. Markedly elevated serum alkaline phosphatase levels were noted in dogs from the high dose group. In addition, there were significantly smaller spleens and elevated liver to body weight ratios in the high dose group. Histopathology was unremarkable in dogs from all dose groups.

Fulfs et al. (1977) conducted a subchronic study in mice, rats and rhesus monkeys. Mice (3-6/sex/group) were fed diets containing 0, 1, 5, 15, or 30 ppm for up to 18 months. Sprague-Dawley rats (4/sex/group) were fed diets containing 0, 5, or 30 ppm for up to 12 months. Monkeys (4/sex/group) received mirex by gavage at 0, 0.25 or 1 mg/kg (equal to 5 and 20 ppm) for up to 26 months. Mice were killed and their livers obtained at 2, 4, 6, 9, 10, 15, and 18 months; rats were killed and surgical biopsies of the livers were taken at 16, 19, 26, and 36 months. Livers from monkeys were obtained and biopsied at 16, 19, 26, and 36 months. In mice, activities of lysosomal β-glycerol phosphatase (ACpase) and glucose 6-phosphatase (G-6-pase) were unchanged at 1 ppm. At higher dose levels, however, G-6-pase decreased in centrilobular areas while ACpase increased with time. In contrast, rat livers had no increase in ACpase and little loss in G-6-pase. Monkey livers showed no loss of G-6-pase or activation of ACpase. Ultrastructurally, livers from treated mice showed an intense proliferation of smooth endoplasmic reticulum that was time- and dose-dependent. Phagocytosis by Kupffer cells of necrotic hepatocytes from mice was observed in the 5, 15, and 30 ppm dose groups. Proliferation of the smooth endoplasmic reticulum from rats and monkeys was less intense than in mice; no other abnormality was noted in hepatocytes from these animals.

4. Chronic Effects

The carcinogenicity of mirex was examined in three experimental animal studies by Innes et al. (1969), Ulland et al. (1977), and NTP (1990). Innes et al. (1969) tested an unspecified commercial mirex formulation in two hybrid strains of mice, (C57B1/6xC3H/AnF)F1 and (C57B1/6xAKR)F1. Mice (18/sex/strain) initially received gavage doses of 10 mg/kg/day on days 7-28 of age and then received a diet containing 26 ppm mirex for 18 months. Untreated and positive controls were included. Mirex produced a significant increase in hepatomas

(hepatoma classification includes both hepatomas and carcinomas combined) in both strains of treated mice (29/65; 65 of 72 animals were necropsied) compared with controls (14/338).

Ulland et al. (1973, 1977) fed groups of Charles River CD rats (26/sex/group) diets containing mirex (99% purity) at 50 or 100 ppm for 18 months. Untreated controls and a positive control (2-AAF) were included. A treatment related decrease in survival was observed. An initial evaluation reported mirex not to be carcinogenic; however, a reevaluation using guidelines for the classification of liver tumors developed at an NCI workshop indicated that it was carcinogenic in the animals under the conditions of study. The only statistically significant tumor increase that could be correlated with mirex exposure was an increase in tumors of the liver (neoplastic nodules). One carcinoma was detected in the low-dose group, five in the high-dose group and none in the control group. This difference was not statistically significant. Only the observation of seven neoplastic nodules of the liver in the high-dose male rats was significant. The fact that this type of nodule has been shown to progress to hepatocellular carcinoma with other carcinogens, the absence of these nodules in the controls, and the fact that this lesion is characteristic of early response to known carcinogens, suggests carcinogenic activity.

The critical study for extrapolation of carcinogenic risk was conducted by the NTP (1990). Groups of 52 F344/N rats of each sex were given diets for 104 weeks containing 0, 0.1, 1.0, 10, 25, or 50 ppm mirex. During the first 6 months of the study, because of the lack of toxicity observed in the females, additional groups of 52 females were given 0, 50, or 100 ppm. Based on feed consumption data, the average intake per day was calculated to be 0, 0.007, 0.075, 0.75, 1.95, and 3.85 mg/kg for male and female rats in the first study and 0, 3.9, and 7.7 mg/kg for female rats in the additional study. Body weights of males in the 25 and 50 ppm dose groups were lower than controls throughout much of the study; body weights of females were comparable to controls except for a 10% - 12% decrease compared to control after week 68 for those animals in the 50 ppm group of the first study. Female rats given 100 ppm in the additional study also had a similar decrease in body weight. Survival was decreased in male rats receiving 25 or 50 ppm only after weeks 86-87 of the study. Survival of treated females was comparable to controls. The most notable effect of mirex administration was on the liver. Incidences of fatty metamorphosis, cytomegaly,

angiectasis (males only), and necrosis of the liver were significantly increased compared to control in male and female rats in the dose groups receiving 10 ppm and above. A dose-related increase in the incidence of neoplastic nodules of the liver was observed; this increase was significantly elevated above the control in the 10, 25, and 50 ppm groups of males and the 50 and 100 ppm groups of females (additional study). Incidences per group were: male: 3/52; 5/52; 5/52; 14/52; 15/52; 26/52 (for dose groups 0, 0.1, 1, 10, 25, 50 ppm, respectively) and female (second study): 2/52; 23/52; 30/52 (for dose groups 0, 50, 100 ppm, respectively). The incidence of neoplastic nodules was not significantly increased in treated females from the first study although the incidence of neoplastic nodules seen in the controls was significantly above historical control incidence. Hepatocellular carcinomas were not significantly increased in any dose group. The incidence of pheochromocytomas of the adrenal gland in male rats was significantly increased compared to control in the 25 and 50 ppm groups (8/51; 7/52; 13/52; 11/52; 18/51; 19/51 for dose groups 0, 0.1, 1, 10, 25, 50 ppm, respectively). There was no compound related increase in pheochromocytomas in treated females. Transitional cell papillomas of the renal pelvis in male rats occurred with a positive trend ($P<0.02$) (0/51; 0/51; 0/52; 0/52; 1/51; 3/52 for dose groups 0, 0.1, 1, 10, 25, 50 ppm, respectively). The incidence of mononuclear cell leukemia showed a dose-related increase in females from the first and additional studies (first study: 8/52; 8/52; 11/52; 14/52; 18/52; 18/52 for dose groups 0, 0.1, 1, 10, 25, 50 ppm, respectively, and additional study: 6/52; 9/52; 14/52 for dose groups 0, 50, 100 ppm, respectively). The incidence of leukemia was significantly increased above control in the 10, 25, 50 and 100 ppm groups when the data are combined.

NTP concluded that "Under the conditions of these 2-year feed studies of mirex, there is *clear evidence of carcinogenic activity* for male and female F344/N rats, as primarily indicated by marked increased incidences of benign neoplastic nodules of the liver, as well as by increased incidences of pheochromocytomas of the adrenal gland and transitional cell papillomas of the kidney in males and by increased incidences of mononuclear cell leukemia in females."

5. Reproductive and Developmental Effects

There are several studies in rats and mice addressing the possible teratogenic and reproductive effects of mirex. Mirex has been found to cause

abnormalities in experimental animal species, including scoliosis, cleft palate, cataracts and heart defects (NAS/NRC 1978). Reproductive effects in animals include reductions in fertility and litter size (Ware and Good 1967; Wole et al. 1979; Gaines and Kimbrough 1970; Chu et al. 1981), decreased birth weight (Chernoff and Kavlock 1982) and reduced survival of offspring (Grabowski and Payne 1980; Grabowski 1982; Gaines and Kimbrough 1970).

Fetal edema has been reported in many of the reproductive studies in laboratory animals (Buelke-Sam et al. 1983; Byrd et al. 1980; Chernoff et al. 1979; Kavlock et al. 1982). Cardiac defects, including dysrhythmias, in fetal and newborn rats have been reported (Grabowski 1982, 1983a,b; Grabowski and Payne 1980). The cardiac defects have been noted at doses as low as 0.1 mg/kg/day for 6-8 days during organogenesis (Grabowski 1983a,b). Using electrocardiography and other functional testing techniques, investigators have reported a significant incidence of cardiovascular and pulmonary effects, in the absence of any visible abnormalities, in neonates born to female rats given mirex at 1 mg/kg/day during gestation (Chernoff et al. 1977). Cataract formation in animals has also been reported by several investigators. Progeny from female rats fed a diet containing mirex (25 mg/kg), before and after mating, had reduced survival rate and a high incidence of cataracts while progeny from females maintained on a diet of 5 mg/kg appeared normal. Further studies demonstrated that the development of cataracts was due primarily to exposure of the neonates to mirex during lactation (Gaines and Kimbrough 1970). Total doses of 10 mg/kg, given singly or over a 4-day period to maternal animals during the first week after delivery, caused cataracts in rats of the CD, Sherman and Long-Evans strains by Day 13, and a 12 mg/kg total dose of mirex resulted in the occurrence of irreversible cataracts which were observed in CD-1 mouse pups after Day 20. In another study, male and female Sprague-Dawley rats fed diets containing 0, 5.0, 10.0, 20.0, or 40.0 ppm mirex for 13 weeks were mated and groups of females were fed the same diet during and after mating and throughout gestation and lactation (Chu et al. 1981). Lesions of the liver and thyroid in the female adults and pups, and cataracts in the eyes of the pups were present from all treated groups.

The effects of mirex on the reproductive performance and behavioral development was determined in a multigeneration study in the prairie vole (Microtus ochrogaster) exposed to mirex in the diet at 0, 0.1, 0.5 0.7, 1.0, or 5.0

ppm (Shannon 1976). At 0.1 ppm (0.015 mg/kg/day), there was a statistically significant decrease in pup survival to 21 days and an increase in pup mortality. The lowest-observed-effect level (LOEL) in this study was 0.1 ppm.

6. Mutagenicity

The mutagenicity of mirex has been examined in both prokaryotic and eukaryotic cells. Mirex was negative in several reverse mutation assays with *Salmonella typhimurium* (Hallett et al. 1978; Schoeny et al. 1979; Rinkus and Legator, 1980; Probst and Hill, 1980; Probst et al. 1981) and similar assays using *Escherichia coli* strains WP2 and WP2 uvrA$^-$ (Probst and Hill 1980; Probst et al. 1981). All assays were conducted with and without metabolic activation. *S. typhimurium*-microsome studies with a preincubation protocol and Salmonella strains TA98, TA100, TA1535, or TA1537 in the presence or absence of metabolic activation showed no mutagenic activity at doses up to 10,000 μg/plate (Mortelmans et al. 1986, NTP 1990). Mirex was also negative in unscheduled DNA synthesis assays (Probst and Hill 1980; Probst et al. 1981; Williams 1980; Maslansky and Williams, 1981; Telang et al. 1981), in experiments on induction of gene mutation at the HGPRT locus in rat hepatocyte mediated cultured human fibroblasts (Tong et al. 1981) and in sister chromatid exchange assays (NTP 1990). A dominant lethal study in Wistar rats was negative (Khera et al. 1976).

D. Toxicity Values

The USEPA has calculated a measure of the carcinogenic potency of mirex (slope factor) of 1.8 $(mg/kg/day)^{-1}$ (USEPA 1990). This value is based on the NTP bioassay (NTP 1990) using Fischer 344/N male rats given mirex in the diet for 104 weeks. Carcinogenicity was indicated by the appearance of liver neoplastic nodules, hepatocellular carinoma, adrenal pheochromocytoma and malignant pheochromocytoma. Mirex is classified by USEPA as a Probable Human Carcinogen B2: sufficient evidence of carcingenicity in animals with inadequate or lack of evidence in humans. It should be noted that the carcinogenicity assessment for mirex is currently under review by the Agency (IRIS 1988).

USEPA has derived a reference dose (RfD) for the noncarcinogenic effects of mirex of 2×10^{-6} mg/kg/day (IRIS 1988). This RfD is based on the vole multi-generation reproduction study of Shannon (1976), in which a LOEL of 0.1 ppm in the diet or 0.015 mg/kg/day was observed, and the application of an uncertainty factor of

10,000. This uncertainty factor consists of an uncertainty factor of 100 to account for intra- and interspecies differences and an additional factor of 100 to account for the fact that a NOEL in the vole reproduction study was not reached, the data were not of chronic duration, and the data base was considered insufficient for determining the most sensitive toxicologic endpoint.

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PHOTOMIREX

A. Summary

Photomirex (8-monohydromirex) is a photodegradation product of the parent compound, mirex. No information on the potential toxicity of photomirex in humans is available. The LD_{50} in rats is reported as approaching 200 mg/kg. The cumulative LD_{50} in mice is 225-250 mg/kg. In subchronic studies in rats, photomirex has been shown to cause mortality, produce hepatomegaly, induce mixed-function oxidases, and produce lesions of the liver, thyroid, and testes. Female animals appear to be less susceptible than males when considering the prevalence and severity of lesions. Photomirex is persistent in tissues and a number of biochemical and histological alterations induced by a subchronic exposure in rats were still present up to 18 months after exposure had ended. Chronic exposure resulted in liver and thyroid toxicity in rats; an increase in thyroid tumor incidence appeared to be treatment related. Photomirex did not produce any visceral or skeletal anomalies in rabbit pups. It did, however, produce decreased litter size, decreased survival indices and cataract formation in rat pups. Photomirex was not mutagenic in a standard Ames assay.

Because of limitations in the available toxicity data for photomirex, the RfD of 2 x 10^{-6} mg/kg/day and the cancer slope factor of 1.8 $(mg/kg/day)^{-1}$ established by USEPA for mirex have been applied to photomirex for purposes of this risk assessment.

B. Health Effects in Humans

No information is available on the potential toxicity of photomirex in humans.

C. Health Effects in Animals

1. Pharmacokinetics

Following administration of ^{14}C-photomirex (42.9 and 4.29 mg/kg) by gavage to Sprague-Dawley rats, absorption was slow (Chu et al. 1979). It was detected in the blood at 1.5 hours and reached its peak concentration at approximately 4 hours after which the concentration rapidly declined. Elimination from the blood after a single intravenous dose (21.5 mg/kg ^{14}C-photomirex) revealed that the semilogarithmic decay of blood concentrations was triphasic. Tissue accumulation of radioactivity after the oral administration was dose-

related; two days after dosing, highest concentrations were found in the fat, followed by liver, skin, thyroid, kidney, heart, testes, spleen, and muscle. Photomirex was exclusively eliminated in the feces. Approximately 38-42% of the dose was excreted in the first 3 days and 51-55% in 28 days. Only trace amounts were detected in urine. No metabolites were detected. Similar results were reported in squirrel monkeys with the exception of the rate of fecal excretion (Chu et al. 1982). Photomirex had a very long half-life in primates. Approximately 10% of an intravenously administered dose was eliminated in feces over a one-year period compared with 51-55% excreted in rat feces in 28 days. In addition, the concentration of photomirex in fat was decreased by 11% in a 36-week period in monkeys in contrast to a 25% reduction of photomirex in the same tissues of rats over a 21-day period.

2. Acute Effects

An acute oral administration of photomirex in mice produced a dose-dependent loss of body weight, however, food consumption was increased or decreased depending on dose (Fujimori et al. 1980). There are no obvious neurotoxic signs and symptoms in mice at various doses, except weakness followed by death. Mortality began to occur 4, 7, and 16 days after daily administration of photomirex at 50, 25, and 10 mg/kg, respectively. The estimated cumulative LD_{50} in mice was 225-250 mg/kg. In another study using mice, the cumulative toxicity (LT_{50}) of 265 mg/kg was reported. In Wistar rats, a dose-response relationship was established for toxicity after a single oral dose of photomirex; 200 mg/kg was estimated as the acute oral LD_{50} (Hallett et al. 1978).

An acute oral exposure of mice to 10 or 25 mg/kg photomirex for four days (Fujimori et al. 1983) and a 15-day exposure of rats to 50 ppm photomirex in the diet (Mehendale et al. 1979) produced an induction of the hepatic mixed function oxidase system.

Exposure of male Sprague-Dawley rats to photomirex at 100 ppm for 15 days (Curtis and Mehendale 1981) or 50 and 150 ppm for 15 days (Curtis et al. 1979) resulted in impaired biliary excretory function. Evidence suggests that hepatobiliary dysfunction may be associated with the inhibition of bile canaliculi-enriched fraction ATPase activities.

3. Subchronic Effects

Photomirex administered in the diet to male or female rats for 28 or 90 days has been shown to produce mortality, body weight gain depression, liver weight changes, biochemical changes, and lesions of the liver, thyroid, and testes (Villeneuve et al. 1979a,b; Singh et al. 1981, Sundaram et al. 1980). The toxic effects produced in the male and female rats are similar, however, there appears to be a sex-related difference in toxicity, with the female being less susceptible than the male.

Male Sprague-Dawley rats (10 animals/group) were fed diets containing 0, 0.5, 5.0, 50.0, or 500 ppm photomirex for 28 days (Villeneuve et al. 1979a). All animals in the highest dose group died after losing approximately 25% of their weight. Clinical signs included irritability, tremors, hypoactivity and a mild cyanosis in the hind feet. At 50 ppm, two animals died. Body weight was depressed in the surviving animals of this group and there was a significant decrease in food intake. Increased liver weight was noted in all groups and the severity was dose-dependent. Lesions of the thyroid and testes were noted at 50 ppm. There were corresponding histopathological alterations in these organs. Increased microsomal enzyme activity and increased serum sorbitol dehydrogenase activity were measured in animals receiving 5.0 ppm or greater. Photomirex accumulated in a dose-dependent manner in all tissues examined, with the highest residues in fat followed by liver, brain, heart, kidney, and spleen.

Alterations in the liver of male weanling rats fed photomirex (0, 0.05, 0.5, 5, or 50 ppm) for 28 days was studied by electron microscopy (Singh et al. 1981). The results indicate that photomirex is hepatotoxic and its effects are dose-dependent. Morphological alterations were detected at all dose levels by electron microscopy.

The effects of a 90-day exposure to photomirex were determined in male rats (10 animals/group) fed the compound for 13 weeks at levels of 0.20, 1.0, 5.0, 25, and 125 ppm in the diet (Villeneuve et al. 1979b). The results confirm the findings in the 28-day study in animals that showed photomirex to be a hepato- and thyrotoxin. At the highest dose, 4 animals died and 1 animal developed cataracts (the animal that developed cataracts was not included in the body weight, food consumption, or histological determinations; it was, however, included in the mortality calculations). Decreased body weight gain and food intake were also observed in high dose animals. Dose-related histological

abnormalities of the thyroid and liver were observed in all treatment groups. Liver weights were increased at 5.0 ppm and higher. Lesions in the testes occurred at the highest dose level. Serum sorbitol dehydrogenase was increased in the highest photomirex group and hepatic aniline hydroxylase activity was increased in a dose-dependent manner at levels of 5 ppm and higher.

The effects of a 28-day and 90-day exposure to photomirex were studied in female rats fed the compound at levels of 0, 0.2, 1.0, 5.0, 25.0, and 125.0 ppm (Sundaram et al. 1980). There were 10 animals/group; half were sacrificed at 28 days, the remainder sacrificed at 90 days. In comparison to the results in the studies using male rats (Singh et al. 1981; Villeneuve et al. 1979a,b), mortality was less in female rats at comparable doses of photomirex. At 125 ppm, 2/5 (40%) female rats died at 28 days and 1/5 (20%) female rats died at 90 days compared to a 50% reported mortality in male rats at 90 days. Food intake and body weight gain were significantly depressed at 125 ppm only at 28 days and not at 90 days in female rats; in the male rats, both parameters were significantly decreased at 90 days. Increased liver weight was reported to be dose-related at 25 and 125 ppm in female rats at both 28 and 90 days; there were no effects on liver weight at 5 ppm and less in female rats. In contrast, liver weight was increased at 5 ppm and higher in male rats. There were no histological alterations of the liver reported at 0.2 ppm in female rats at 28 and 90 days; liver lesions were dose-dependent at 1 ppm and higher. In male rats, histological liver lesions were detected at the lowest dose of 0.2 ppm. In the thyroid, no histological effects were observed in female rats at or below 1.0 ppm at 28 days and at 0.2 ppm at 90 days. Although thyroid lesions were detected at 0.2 ppm in male rats at 90 days, the incidence of lesions at succeeding dose levels was not consistently higher in males than females, therefore, a sex-related susceptiblity for thyroid injury was not as apparent as for liver damage. Histological changes of the reproductive organs were absent at all dose levels in females.

The toxicological changes produced after a 28-day treatment with photomirex have been shown to persist when measured at 48 weeks and 18 months after the cessation of exposure (Chu et al. 1981a). When weanling male Sprague-Dawley rats were given diets containing 0, 0.05, 0.5, 5.0, or 50 ppm photomirex for 28 days and given a clean diet thereafter, liver hypertrophy persisted in the highest dose group for 24 weeks. Elevated serum sorbitol dehydrogenase activity persisted in this group for 12 weeks. Histological changes

in liver and thyroid were present up to 48 weeks in the 5.0 and 50 ppm groups. Testicular changes were not evident after 12 weeks. Residue data showed that next to fat, liver contained the highest concentrations of photomirex, even at 48 weeks post exposure. A study in weanling male Sprague-Dawley rats using the same concentrations of photomirex showed alterations by light and electron microscopy in the thyroid which persisted at least 18 months after a 28-day exposure (Singh et al. 1982).

4. Chronic Effects

Chronic exposure to photomirex in rats also produces hepato- and thyrotoxicity. Male weanling Sprague-Dawley rats (10 animals/group) given diets containing 0, 0.2, 1.0, 5.0, 25, and 125 ppm for 21 months had treatment-related histological lesions in the livers and thyroids of rats from all dose groups (Chu et al. 1981c). All animals in the 125 ppm group died during the study. Clinical signs of toxicity included hypoactivity, irritability, and cyanosis of the hindlimbs. Weight loss and reduced food consumption were noted at the high dose. Increased liver weight was noted in groups given 5.0 ppm photomirex and higher. Serum sorbital dehydrogenase activity was increased in groups given 25 ppm and higher. Hepatic microsomal aniline hydroxylase activity was increased in the 1.0 and 25 ppm groups. A dose-dependent accumulation of photomirex was noted in the rats, with highest levels detected in fat, followed by liver, testes, brain, spleen, heart and kidney. The residue levels in fat were higher than those measured in the 90-day experiment, indicating that a storage equilibrium had not been achieved in 90 days. The occurrence of thyroid tumors appeared to be increased and related to treatment in animals from the 25 ppm group (4/10) compared to control (1/10). No thyroid tumors were observed in animals from the other dose groups.

5. Reproductive and Developmental Effects

A teratogenicity study in the New Zealand white rabbit used single daily oral doses of 0, 5, or 10 mg photomirex/kg from the 6th to the 18th day of gestation (Villeneuve et al. 1979c). There was no maternal toxicity. There was a significant reduction in the mean fetal weight of the 10 mg/kg group, however, all other indices of fetal survival and development were within control range. Photomirex appeared to readily cross the placenta in rabbits and accumulated in a dose-dependent fashion; it was found at the highest levels in the fetal heart,

followed by liver, brain and blood. There were no teratologic effects at the doses used.

Reproductive impairment was reported in a study of photomirex in the rat; groups of weanling male and female rats were fed diets containing the chemical at levels of 0, 2.5, 5.0, 10, 20, or 40 ppm for 91 days prior to mating, 15 days during mating, and throughout gestation and lactation (Chu et al. 1981b). At 40 ppm photomirex there was a significant decrease in maternal weight gain. A decreased incidence of females showing sperm in vaginal smears was detected at 5.0 ppm. Litter size was decreased in all treatment groups, however, survival indices of pups were affected only at 40 ppm. There was a dose-dependent accumulation of photomirex in the liver of rat pups. Histopathology revealed lesions in the liver and thyroid of the female adults and pups, and in the eyes of the pups. Cataracts were observed in the pups of the treated groups starting from the third week of nursing.

6. Mutagenicity

Photomirex was not mutagenic in a standard Ames assay with liver microsomal activation using *Salmonella typhimurium* strains TA1535, TA100, TA1537, TA1538, and TA98 (Hallett et al. 1978).

D. Toxicity Values

Currently, there is no appropriate study of photomirex from which to calculate an estimate of carcinogenic potency. However, in the study by Chu et al. (1981c), an increased incidence in thyroid tumors occurred in rats at 25 ppm photomirex, which is comparable to the dose levels at which increased tumor incidence was noted with mirex. Therefore, the USEPA calculated cancer potency estimate (or slope factor) for mirex of 1.8 $(mg/kg/day)^{-1}$ will also be used as a conservative estimate of the carcinogenic potency of photomirex.

Currently, there is no appropriate study of photomirex from which to calculate an RfD. The chronic study by Chu et al. (1981c) in the rat yielded a high incidence of liver and thyroid lesions even at the lowest dose tested. It is unclear from this study at what dose level a no-observed-effect level (NOEL) would be achieved. In additional studies of subchronic duration, there were no NOELs reported even at the lowest doses tested for male rats. It would be difficult to assign a safety factor to account for the fact that a NOEL for these endpoints had not been reached. However, the USEPA RfD for mirex

is based on a reproduction study and a comparative reproduction study by Chu et al. (1981b) indicates that mirex and photomirex are similar in toxicity with respect to this endpoint. Therefore, the RfD of 2×10^{-6} mg/kg/day estimated for the noncarcinogenic effects of mirex will be used as a conservative estimate of the RfD for photomirex.

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APPENDIX C
Ohio Department of Health Survey

246 N. High Street
Post Office Box 118
Columbus, Ohio 43266-0118

Telephone (614) 466-3543

RICHARD F. CELESTE
Governor

Dear Resident;

The Ohio Department of Health (ODH) is attempting to find out how many people in the Salem to Lisbon area have had exposure to the pesticide Mirex, either through the contaminated Middle Fork of the Little Beaver Creek or from the Nease Chemical Company. Mirex is classified as a potential human carcinogen. ODH and the Ohio Environmental Protection Agency are concerned about people who regularly used the upstream part of the creek because the sediments and fish are contaminated with pesticides. The Nease Chemical Company operated a plant just outside of Salem from 1961 until the EPA closed it in 1973. We are also concerned that former employees of the Nease Chemical Company may have been exposed to high levels of Mirex.

Many citizens in the area have expressed health concerns related to the pollution of the Middle Fork of the Little Beaver Creek and the Nease Chemical Company. Before we investigate any possible health problems, it is first very important to find out how many people were potentially exposed to Mirex in the creek or while working for the Nease Chemical Company. This survey is designed to assess how many people may have been exposed and for how long.

In order that the results truly represent the exposure level of the people living in the Middle Fork of the Little Beaver Creek area, it is important that each survey be completed and returned. We would like the questionnaire to be filled out by an adult member of your household, but include information about all members of the household. Please return the completed survey in the enclosed stamped return envelope as soon as possible.

Results of this survey will be released only in aggregate form. Your identity will be kept confidential. The first page of the survey containing your name and address will be separated from the remainder of the form and used only to identify who has completed the survey.

The results of this research will be made available to officials and representatives in the state and local government, members of Congress, the Ohio EPA, the U.S. EPA, the Ruetgers-Nease Company, and all interested citizens.

Your contribution to this effort is greatly appreciated. We would be happy to answer any questions you might have. Please write or call Mary Rouse in the Division of Epidemiology and Toxicology. The toll-free telephone number is 1-800-282-0546.

Sincerely,

Deborah Gray
Chief, Toxicology Branch

DLG/mar

MIDDLE FORK OF THE LITTLE BEAVER CREEK
COLUMBIANA AND MAHONING COUNTIES
OHIO DEPARTMENT OF HEALTH SURVEY

1. Please list all of the people who have lived in your household since 1961 by first and last name, sex, date of birth, and years of residence at the current address:

Name (first and last)	Sex (M/F)	Date of Birth (mo/day/yr)	Years of Residence (ex: 1961-1980)

(If you need more space, please attach another piece of paper.)

2. Please list your current address:

_____

_____

home phone number: _____

daytime phone number: _____
(if different from home)

3. Please list your former address within the Salem-Lisbon area and years of residence (if any):

Address	Years of Residence

The above information will be kept strictly confidential and separate from the rest of the survey. It is needed to identify who has completed the survey. For the remainder of the questionnaire, please identify household members by age and sex only (for example: female, 52 years old).

4. Please list present employer and all former employers since 1960 of all adult residents of this household listed in question 1, along with the dates of employment and their current age.

Employee's present age and sex Employer Dates of Employment

_____

_____

_____

_____

_____

_____

_____

5. Did you know that the Ohio Department of Health issued a fish consumption advisory for the Middle Fork of the Little Beaver Creek between Salem and Lisbon in October 1987? (circle)

1 NO
2 YES

6. From 1961 until the fish advisory was issued in October 1987, did you or anyone in your household eat fish caught from the Middle Fork of the Little Beaver Creek between Salem and Lisbon?

1 NO ---------->IF NO, SKIP FROM HERE
<-------- 2 YES TO QUESTION 10

(IF YES)

7. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER(SPECIFY):_____

8. Using the map on page 8, put "8" by the location(s) that was most frequently fished.

9. What type of fish from this area was most often consumed?

_____

10. Since the fish advisory was issued in October 1987, have you or anyone in your household eaten fish caught from the Middle Fork of the Little Beaver Creek between Salem and Lisbon?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 14

(IF YES)

11. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER(SPECIFY):_____

12. Using the map on page 8, put "12" by the location(s) that is most frequently fished.

13. What type of fish from this area is most often consumed?

_____

14. Since 1961, have you or any member of your household eaten game other than fish (such as deer or rabbit) hunted or trapped from the Columbiana/Mahoning County area near the Middle Fork of the Little Beaver Creek between Salem and Lisbon?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 18

(IF YES)

15. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER(SPECIFY):_____

16. What type of game from this area is most often consumed?

_____

17. Where is the location most frequently used for hunting or trapping? If possible, use the map on page 8 and put "17" by the location(s) that is most frequently used to hunt or trap.

_____

18. Did you know that the Ohio Department of Health issued a contact advisory warning against swimming, wading, etc, for the Middle Fork of the Little Beaver Creek between Salem and Lisbon in March 1988?

1 NO
2 YES

19. From 1961 until the contact advisory was issued in March 1988, did you or members of your household swim, wade or play in the Middle Fork of the Little Beaver Creek anywhere between Salem and Lisbon?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 22

(IF YES)

20. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER(SPECIFY):_____

21. Using the map on page 8, put "21" by the location(s) that was most frequently used.

22. Since the contact advisory was issued in March 1988, have you or any member of your household been swimming, wading, or playing in the Middle Fork of the Little Beaver Creek anywhere between Salem and Lisbon?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 25

(IF YES)

23. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER (SPECIFY):_____

24. Using the map on page 8, put "24" by the location(s) that is most frequently used.

25. Do you live on a farm near the creek?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 31

(IF YES)

26. Do you use water from the Middle Fork of the Little Beaver Creek for irrigation?

1 NO
2 YES

27. Are any of the fields or pastures on your farm on the flood plain of MFLBC?

1 NO
2 YES

28. Are any animal or vegetable products from your farm consumed?

1 NO ---------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 31

(IF YES)

29. What type of animal and/or vegetable products from your farm are most often consumed?

_____

30. How often are any animal or vegetable products from your farm consumed?

1 ONCE OR MORE A DAY
2 THREE TO FOUR TIMES A WEEK
3 ONCE OR TWICE A WEEK
4 ONCE OR TWICE A MONTH
5 ONCE OR TWICE EVERY SIX MONTHS
6 ONCE A YEAR OR LESS
7 OTHER (SPECIFY):_____

31. Do you or anyone in your household ever eat fruit or vegetables grown in your garden or a garden in the area of the Middle Fork of the Little Beaver Creek between Salem and Lisbon?

1 NO ---------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 35

(IF YES)

32. Is water from the Middle Fork of the Little Beaver Creek used for irrigation in the garden?

1 NO
2 YES

33. What types of fruits or vegetables from that garden are most often consumed?

_____

34. How often do are any fruits or vegetables from that garden consumed?

1 ONCE A DAY OR MORE
2 THREE TO FOUR TIMES A WEEK
3 ONCE OR TWICE A WEEK
4 ONCE OR TWICE A MONTH
5 ONCE OR TWICE EVERY SIX MONTHS
6 ONCE OR LESS A YEAR
7 OTHER (SPECIFY):_____

35. What is the source of the water that comes into your home for drinking, bathing, etc?

1 CITY SUPPLY
WHAT CITY?_____
2 DUG WELL
3 DRILLED WELL
4 OTHER (SPECIFY):_____

36. Have you ever used the Middle Fork of the Little Beaver Creek or its water for anything else not already covered in this survey, such as dredging or other work-related activities?

1 NO
2 YES (SPECIFY):_____

37. Approximately how close do you live to the nearest part of the Middle Fork of the Little Beaver Creek?

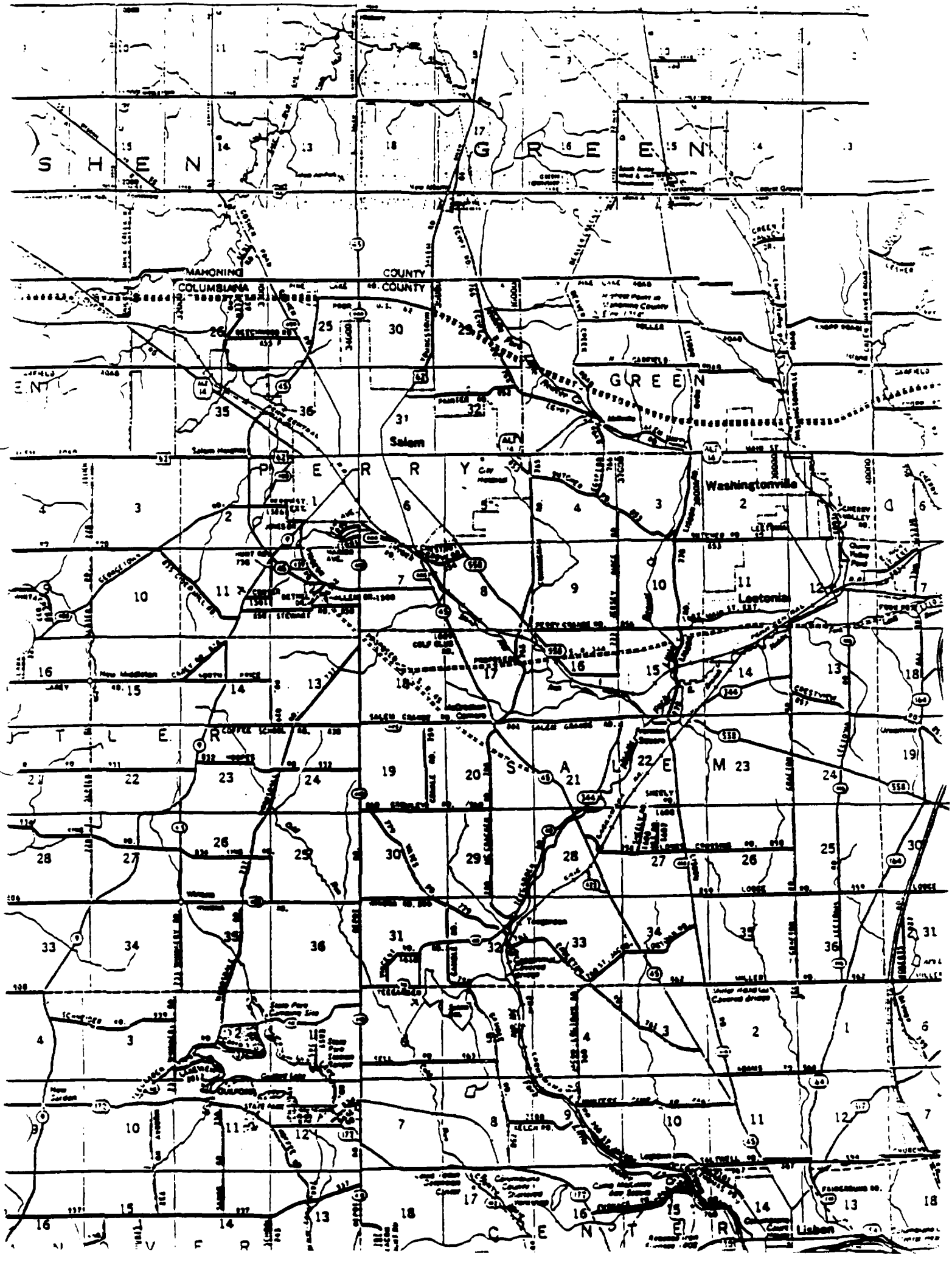
1 LIVE ON THE BANKS OR FLOODPLAIN
2 A QUARTER OF A MILE OR LESS
3 ONE QUARTER TO ONE HALF OF A MILE
4 ONE HALF TO ONE MILE
5 ONE TO TWO MILES
6 MORE THAN TWO MILES

38. Using the map on page 8, put an "X" at the location where you live

Is there anything else you would like to tell us regarding the Middl Fork of the Little Beaver Creek, the Nease Chemical Company, or an possible health conditions you feel may be related to the above? If so please use this space and the back of this sheet as needed.

Also, any comments you wish to make that you think may help us i future efforts to better understand the situation will be appreciated either here or in a separate letter.

MAR/mar
8/89


S H E N
G R E E N
MAHONING COUNTY
COLUMBIANA CO. COUNTY
G R E E N
Salem
P E R R Y
Washingtonville
Lestonia
T L E R
S A L E M
C E N T E R
Lisbon

INTEROFFICE COMMUNICATION

To: Rob Winters, Environ Corporation
From: Mary Rouse Martin, Ohio Department of Health
Date: February 13, 1991
Subject: Ruetgers-Nease Superfund Site Survey Data

Rob,

Please find attached a copy of the results of the survey conducted by the Ohio Department of Health, Bureau of Epidemiology and Toxicology in September 1989 regarding potential exposure to contaminants from the Ruetgers-Nease Superfund Site. This information was reported in condensed form in "Assessment of Exposure to Mirex Associated with the Nease Chemical Company Superfund Site in Salem, Columbiana County, Ohio" from the Ohio Department of Health, October 4, 1990. The tables summarize the information regarding the five potential pathways for exposure under study by the Ohio Department of Health. Please note that the results for the fish consumption and sediment contact are for the period of time since the advisories were issued in 1987 and 1988, respectively.

If can be of further assistance, please give me a call.

Sincerely,
Mary Rouse Martin

Mary Rouse Martin
Epidemiologist
Bureau of Epidemiology and Toxicology
Ohio Department of Health
P.O. Box 118
Columbus, Ohio 43266-0118
614/644-6447

APPENDIX H: FREQUENCY OF ACTIVITIES RELATED TO THE NEASE SUPERFUND SITE AND MFLBC AS REPORTED AMONG 200 SURVEY RESPONDENTS

Table I: Frequency of Contact with MFLBC and its Sediments Among 200 Survey Respondents

contact frequency	number of respondents	percentage
none	119	59.5
<1/year	16	8.0
1/year	7	3.5
1-2/6 months	21	10.5
1/month	12	6.0
≥1/week	25	12.5

Table II: Frequency of Consuming Game Hunted or Trapped from MFLBC Area Among 200 Survey Respondents

consumption frequency	number of respondents	percentage
none	135	67.5%
>1/year	12	6.0%
1/year	17	8.5%
1-2/6 months	22	11.0%
1/month	10	5.0%
≥1/week	4	2.0%

Table III: Frequency of Consuming Fish from MFLBC Among 200 Survey Respondents

consumption frequency	number of respondents	percentage
none	166	83.0%
>1/year	6	3.0%
1/year	4	2.0%
1-2/6 months	11	5.5%
1/month	6	3.0%
≥1/week	7	3.5%

Table IV: Frequency of Consumption of Garden Products Irrigated by MFLBC Water for Crop Irrigation Among 200 Survey Respondents

consumption frequency	number of respondents	percentage
none	183	91.5%
1/year	1	0.5%
1-2/6 months	2	1.0%
1-2/month	2	1.0%
1-2/week	3	1.5%
3-4/week	9	4.5%

From: Rouse Martin M, Shelley TL, Mortensen BK. Assessment of Exposure to Mirex Associated with the Nease Chemical Company Superfund Site in Salem, Columbiana County, Ohio. Ohio Department of Health. October 4, 1990.

Table V: Frequency and Duration of Employment Related to Possible Mirex Exposure Among 200 Survey Respondents

type of employment	number of respondents	percentage
not related to mirex	189	94.5%
Nease/possible contact	11	5.5%

Table VI: Frequency of Consumption of Products from Animals with Access to MFLBC Among 200 Survey Respondents

consumption frequency	number of respondents	percentage
none	191	95.5%
≤1/year	1	0.5%
1-2/6 months	0	0.05
1-2/month	2	1.0%
1-2/week	0	0.0%
≥3-4/week	6	3.0%

From: Rouse Martin M, Shelley TL, Mortensen BK. Assessment of Exposure to Mirex Associated with the Nease Chemical Company Superfund Site in Salem, Columbiana County, Ohio. Ohio Department of Health. October 4, 1990.

APPENDIX D
Exposure Assumptions

EXPOSURE ASSUMPTIONS

A. Ingestion of Soil (TABLE 19)

Ingestion Rate (IR)

The soil ingestion rates of 200 mg/day for children ages 1 to 6 and 100 mg/day for older age groups are recommended by USEPA (1989). Recent investigations of soil ingestion by children (Calabrese et al. 1989; Davis et al. 1990; van Wijnen et al. 1990) suggest that soil ingestion rates are lower than estimated by USEPA.

The Calabrese et al. (1989) study used an approach for estimating soil ingestion rates in young children based on measurement and mass-balance of eight tracer elements in children's stools, taking into consideration background contributions from tracer elements found in food and medicine. Calabrese et al. reported that only three of the eight tracers (aluminum, silicon and yttrium) provided a reliable basis for estimating soil ingestion. Median estimates of soil ingestion by children from these tracers ranged from 9 mg/day to 40 mg/day.

Davis et al. (1990) used a soil tracer methodology similar to Calabrese et al. (1989) to assess the daily soil ingestion of 104 children between the ages of two and seven years in a three-city area in southeastern Washington State. The investigators used aluminum, silicon, and titanium as tracer elements and a mass-balance approach similar to that of Calabrese et al. to account for all intake (food and nonfood) and output (feces and urine). Contributions of tracer elements found in food and medicines were subtracted in estimating daily soil ingestion. The median daily soil ingestion rates for the three tracers ranged from 25.3 to 81.3 mg/day.

The study by van Wijnen et al. (1990) measured aluminum, titanium and acid-insoluble residue (AIR) in soil and feces from over four hundred children in the Netherlands in three different environmental situations: day-care centers, campgrounds, and hospitals. To account for intake of these tracers from sources other than soil, the amount of soil ingested was estimated to be not higher than the lowest of the three separate estimates. The estimate, referred to as the limiting tracer method (LTM) value, was then corrected for the mean LTM value calculated similarly for the group of hospitalized children without access to soil and dust. The authors concluded that the geometric mean of the amount of soil ingested by young children under "normal living

conditions" based on estimates of day-care center groups is between 0 to 90 mg/day and that 190 mg/day is the 90th percentile estimate.

The results of these three soil ingestion studies suggest that an average soil ingestion rate for young children is about 50 mg/day. Therefore, the soil intakes derived in this assessment using USEPA values are likely to be overestimates of exposure.

Fraction Ingested from Contaminated Source (FI)

Because soil contamination is limited to the flood plain and because individuals' activities take place over a variety of locations, it is reasonable to assume that not all soil contact is with flood plain soil. For the purposes of this assessment, the conservative assumption was made that 50% of the soil ingested was flood plain soil.

Bioavailability Factor (BF)

The bioavailability of chemicals in a soil matrix is chemical-specific and is also influenced by soil properties. In the absence of chemical-specific data, the ingestion bioavailability factor is assumed to be 100%.

Exposure Frequency (EF)

The USEPA (1989) recommended RME exposure frequency of 365 days/year was adopted for purposes of this assessment.

B. Ingestion of Sediment (TABLES 20 and 21)

Ingestion Rate (IR)

See Ingestion of Soil - Ingestion Rate

Fraction Ingested from Contaminated Source (FI)

Ingestion of contaminated sediment would only occur on those days when an individual spent time along the MFLBC. The assumption was made that 50% of the amount of soil/sediment ingested on those days was contaminated sediment from the MFLBC.

NO
Should also be 100% or higher adjust the EF but, assume all soil ingested was contaminated

Bioavailability Factor (BF)

See Ingestion of Soil - Bioavailability Factor

Exposure Frequency (EF)

The exposure frequency for ingestion of sediment is the same as that for dermal contact with sediment. See Section D below.

C. Dermal Contact with Soil (TABLE 22)

Absorption Factor (ABS)

Dermal absorption of chemicals from soil is a function of the degree of skin contact, the extent of desorption of the chemical from soil, and the rate at which a chemical penetrates the skin and enters the blood stream. Because undamaged skin provides a fairly effective barrier against the absorption of soil-bound chemicals, dermal absorption of chemicals from soil will be less than 100%. Few chemicals have been tested experimentally for dermal absorption from soil, however, and most of the available data comes from studies of pure compounds or chemicals in solution.

Consideration of [redacted]

The relative dermal absorption factors for the chemicals of potential concern derived for purposes of this risk assessment are presented in Chapter IV of this report.

So what is the dermal abs factor? Restate these 1% is low - recheck SB 570 any

Adherence Factor (AF)

Several studies have measured soil adherence to skin either directly (Lepow et al. 1975) or indirectly (Roels et al. 1980; CDHS 1986; Que Hee et al. 1985). A recent study by Driver et al. (1989) measured the adherence (mg soil per square centimeter of skin surface area, mg/cm^2) of 5 different soil types. Both sieved and unsieved soils were examined, and results were reported according to soil type, organic content, and particle size. Soil adherence values ranged from 0.17 to 0.90 mg/cm^2 for unsieved soil. In the absence of site-specific information on soil type, a soil adherence factor of 0.9 mg/cm^2 was conservatively used.

1.0 mg/cm2 is recommended p2-16 of Interim Guidance for Dermal expos. Assmt.

Exposure Frequency (EF)

Exposure frequencies for dermal contact with soil are based on estimates of the number of days individuals might come in contact with soil outside their residence (e.g., through gardening, playing, etc.). The reasonable maximum exposure frequency for

children is based on guidance in USEPA (1989, p. 6-43), which recommends that children are outdoors 3 times per week in the fall and spring (when temperatures are above 32°F) and 5 times per week in the summer. From this guidance and knowledge of climate data for the Youngstown, Ohio area (the temperature is above freezing 230 days per year, i.e., approximately 35 weeks or 8 months; U.S. Department of Commerce 1985) a value of 131 days/year of potential contact with soil for the reasonable maximum exposure can be estimated. This value is based on an exposure frequency of 5 times/week for 13 weeks in the summer and 3 times/week for 22 weeks in the spring and fall when temperatures are above 32°F. Because time spent outdoors by children is likely to include a variety of activities over a number of locations, only some of the time that children are estimated to spend outdoors is expected to result in any contact with contaminated soil at their residence. The exposure frequency for children used here is, therefore, considered to be a conservative estimate.

Information on the number of days spent outside by adults is unavailable; however, it can be assumed that adults are outside less frequently than children. For the purposes of this assessment, the assumption is made that adults are potentially exposed 2 days per week for the 8 months of the year (35 weeks) that the temperature is above freezing or 70 days per year.

Fraction Contacted from Contaminated Source (FC)

Because soil contamination is limited to the flood plain and because individuals' activities take place over a variety of locations, it is reasonable to assume not all soil contact is with flood plain soil. For the purposes of this assessment, the conservative assumption was made that 50% of the soil contacted was flood plain soil.

Skin Surface Area (SA)

The amount of skin available for exposure to soil is a function of such factors as weather, expected activities, and types of clothing worn by individuals. Because human activity patterns are highly variable, estimates of the amount of skin in contact with soil have been based on a series of plausible (but conservative) assumptions about the type of dress and activities of floodplain residents along the MFLBC. Specifically, the assumption was made for this population that short-sleeved shirts and shorts are worn during warm-weather months (estimated to be one-half the amount of time spent outdoors) and long-sleeved shirts or jackets and long pants are worn during cool-weather months (the remaining half of the time spent outdoors). Accordingly, the area of

potential dermal contact with soil was assumed to be an average of the surface area potentially exposed during warm weather (i.e., face (approximated as one-half the head), two-thirds of the upper extremities and one-half of the lower extremities) and the surface area potentially exposed during cool weather (i.e., one-half the head and the hands).

For children, surface areas of the potentially exposed body parts (hands, one-half the head, upper limbs, and lower limbs and feet) were derived by multiplying the percentage of total body surface area each body part comprises by total body surface area. Total body surface area values for children were determined by averaging male and female 50th percentile total body surface area values from Tables 4B-3 and 4B-4 in the Exposure Factors Handbook (USEPA 1990). The total body surface area value for 1<2 year old children is the value listed in ICRP (1984) for 2 year olds.

The percentages of total body surface area by body part were derived from Table 4-3 of the Exposure Factors Handbook (USEPA 1990). The percent surface area values for arms and hands were used to estimate the percent surface area of the upper extremities. The percent surface area values of the lower extremities were the combined values of the legs and feet. Because body surface area data are limited, the percent surface area values for the age groups modeled in this risk assessment did not include values for all ages within the age group. The values for average percentage of body surface area for each group were derived by averaging the surface area values for the age groups as shown below.

Age Group	Age Group Data Source (Exposure Factors Handbook)	SA (cm^2/day)
1>6	1<2, 2<3, 3<4, 4<5	1606
6>12	6<7, 9<10	2459
12>18	12<13, 13<14, 16<17, 17<18	3650

5,000 cm^2 for adults, 2500 for children

For adults (>18 years), percentages of total body surface area of potentially exposed body parts were derived by averaging values for men and women from Table 4-2 of the Exposure Factors Handbook. These values were multiplied by the mean total body surface area for men and women (from Table 4-1) to obtain a value of 4019 cm^2/day for skin surface area exposed.

D. Dermal Contact with Sediment (TABLES 23 and 24)

Absorption Factor (ABS)

See Dermal Contact with Soil - Absorption Factor.

Adherence Factor (AF)

See Dermal Contact with Soil - Adherence Factor.

Exposure Frequency (EF)

The frequency of exposure to sediment currently in the area downstream of the advisory for the MFLBC and in the future along sections of the creek both within the advisory area and downstream of the advisory area is assumed to be the same as that for residential contact with soil, with one exception. Children from the ages of 1 to 6 are unlikely to play along the MFLBC by themselves. Rather, they would more likely be accompanied by an adult. Thus, their exposure frequency would be equal to that derived for an adult, or 70 days per year.

Due to the advisory posted along the MFLBC, the frequency of exposure to sediment upstream of Lisbon Dam is assumed to be one-half that for current downstream and future upstream and downstream exposure, or 35 days, 66 days, and 35 days for adults, 15 and 9 year olds, and 4 year olds, respectively. This is a reasonably conservative estimate of frequency of contact with the sediment based on the results of a survey conducted in September 1989 by the Ohio Department of Health (ODH 1990). The survey results showed only 12.5 percent of the respondents were in contact with the MFLBC once per week or more and 87.5 percent had contact with the MFLBC once per month or less. The ODH survey results are included in Appendix C.

Skin Surface Area (SA)

See Dermal Contact with Soil - Skin Surface Area.

E. Ingestion of Beef (TABLE 25)

Ingestion Rate (IR)

An average beef ingestion rate for adults of 0.1 kg/day is suggested by USEPA (1990) and has been adopted for purposes of this assessment.

In the absence of USEPA guidance, age-specific ingestion rates were calculated using data gathered by Pao et al. (1982). For adults, the value of 0.1 kg/day recommended by USEPA corresponds most closely to the 75th percentile beef consumption value reported by Pao et al. (1982). As a result, 75th percentile consumption values were used to derive age-specific beef ingestion rates. The age groups presented in Pao et al. do not correspond directly to the age groups modeled in this assessment. The values for beef ingestion were calculated by averaging the ingestion values for the age groups as shown below:

Age Group	Age Group Data Source (Pao et al. 1982)	IR (kg/day)
1>6	1-2, 3-5	0.049
6>12	6-8, 9-14	0.074
12>18	9-14, 15-18	0.095

Fraction Ingested from Contaminated Source (FI)

USEPA (1990) guidance recommends for the reasonable maximum case that it be assumed that 75 percent of an individual's daily intake of beef would be homegrown. This assumption was adopted for purposes of this assessment.

Exposure Frequency

An exposure frequency of 365 days/year was assumed based on USEPA guidance (USEPA 1990).

F. Ingestion of Milk (TABLE 26)

Ingestion Rate (IR)

An average milk ingestion rate for adults of 0.305 kg/day is reported by USEPA (1990) and has been adopted for purposes of this assessment.

In the absence of USEPA guidance, age-specific ingestion rates were calculated using data gathered by Pao et al. (1982). For adults, the value of 0.305 kg/day cited by USEPA corresponds most closely to the 75th percentile whole fluid milk consumption value reported by Pao et al. (1982). As a result, 75th percentile consumption values were used to derive age-specific milk ingestion rates. The age groups presented in Pao et al. do not correspond directly to the age groups modeled in this assessment. The

values for milk ingestion were calculated by averaging the ingestion values for the age groups as shown below:

Age Group	Age Group Data Source (Pao et al. 1982)	IR (kg/day)
1>6	1-2, 3-5	0.509
6>12	6-8, 9-14	0.529
12>18	9-14, 15-18	0.519

Fraction Ingested from Contaminated Source (FI)

USEPA (1990) guidance recommends for the reasonable maximum case that it be assumed that 75 percent of an individual's daily intake of milk would be from homegrown cattle. This assumption was adopted for purposes of this assessment.

Exposure Frequency

An exposure frequency of 365 days/year was assumed based on USEPA guidance (USEPA 1990).

G. Ingestion of Homegrown Vegetables (TABLE 27)

Ingestion Rate (IR)

Age-specific average ingestion rates for vegetables were obtained from data reported by the USDA (1983). The USDA average ingestion rate for adults is consistent with the recommended USEPA value. As a result, the USDA ingestion rate for adults and the corresponding age-specific values have been adopted for purposes of this assessment. The age groups reported in USDA (1983) do not correspond directly to the age groups modeled in this assessment. The values for vegetable ingestion were calculated by averaging the ingestion values for the age groups as shown below:

Age Group	Age Group Data Source (USDA 1983)	IR (kg/day)
1<6	1-2, 3-5	0.104
6<12	6-8, 9-11	0.155
12<18	12-14, 15-18	0.182
>18	19-22, 23-34, 35-50, 51-64, 65-74	0.224

Fraction Ingested from Contaminated Source (FI)

USEPA (1990) guidance recommends for the reasonable maximum case that it be assumed that 40 percent of an individual's daily intake of vegetables would be homegrown. This assumption was adopted for purposes of this assessment.

Exposure Frequency

An exposure frequency of 183 days/year (i.e., 50% of the year) was assumed based on USEPA (1990) guidance for the reasonable maximum exposure case.

H. Ingestion of Fish (TABLES 28 and 29)

Ingestion Rate (IR)

USEPA (1990) recommends an adult fish ingestion rate of 0.1 to 0.2 kg/meal. ENVIRON has assumed that 0.15 kg/meal represents a typical ingestion rate and has adopted that value for the purposes of this assessment.

In the absence of USEPA guidance, age-specific ingestion rates were calculated using data gathered by Pao et al. (1982). Based on the 50th percentile average daily fish consumption values reported by Pao et al., it was determined that children ages 1 to 6 consume 47% of what an adult consumes, children ages 6 to 12 consume 61% of what an adult consumes and children ages 12 to 18 consume 70% of what an adult consumes. The age groups presented in Pao et al. do not correspond directly to the age groups modeled in this assessment. These percentages where calculated by averaging the data for each age group as shown below. These percentages were multiplied by 0.15 kg to estimate the reasonable maximum amount ingested per meal for each age group.

Age Group	Age Group Data Source (Pao et al. 1982)	% of adult	IR (kg/day)
1>6	1-2, 3-5	47	0.071
6>12	6-8, 9-14	61	0.092
12>18	9-14, 15-18	70	0.105
>18	19-34, 35-64, 65-74	100	0.150

Fraction Ingested from Contaminated Source (FI)

An FI of 1 is assumed because the scenario models the consumption of fish from the MFLBC only.

Exposure Frequency (EF)

Exposure frequency values for current exposure in the area of the fishing advisory (i.e., upstream of Lisbon Dam) via fish ingestion are based on data collected in September 1989 by the ODH (1990). Eight-three percent of the respondents reported that they do not consume any fish caught in the MFLBC. The 93.5th percentile of the respondents reported eating fish caught from the MFLBC approximately one or two times in a six month period or less. Consequently, an exposure frequency of 1 to 2 days/6 months or 4 days/year approximates the 90th to 95th percentile exposure frequency and was adopted for the purposes of this assessment. The ODH survey results are included in Appendix C.

It was assumed that the recreational populations would fish more often in the area downstream of the advisory. A future exposure scenario with a higher exposure frequency was also developed for the advisory area because USEPA guidance requires that hypothetical exposures be calculated as if no advisory were in place. For the purpose of this assessment, therefore, the reasonable maximum exposure frequency was assumed to be 1 day/week or 52 days/year for ingestion of fish caught downstream of Lisbon Dam under current conditions and in the future both upstream and downstream.

I. Ingestion of Game (TABLE 30)

Ingestion Rate (IR)

The ODH survey provides information on the frequency of consumption of game in terms of number of meals consumed over various time periods. These data are similar in nature to the data provided in this survey for consumption of fish from the MFLBC. In the absence of standard assumptions about the amount of game eaten per meal, it was assumed this value was equivalent to the amount of locally-caught fish consumed per meal.

Fraction Ingested from Contaminated Source (FI)

It was conservatively assumed that all of the game consumed by local hunters was taken from the area along the MFLBC. The FI value was therefore assumed to be 1.

Exposure Frequency (EF)

Exposure frequency values for current and future exposure via ingestion of game are based on data collected in September 1989 by the Ohio Department of Health (ODH

1990). Ninety-eight percent of the respondents to the ODH survey reported eating game caught in the area surrounding the MFLBC approximately once per month or less. Consequently, an exposure frequency of 1 day/month or 12 days/year approximates the 95th percentile exposure frequency and was adopted for the purposes of this assessment. The ODH survey results are included in Appendix C.

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dgm\0439c\appx2

APPENDIX E
Cancer and Noncancer Risk Tables

FLOOD PLAIN RESIDENT

FLOOD PLAIN RESIDENT: CURRENT & FUTURE USE

CHEMICAL	Soil Ingestion Adult CANCER RISK	Soil Ingestion Fifteen year old CANCER RISK	Soil Ingestion Nine year old CANCER RISK	Soil Ingestion Four year old CANCER RISK	Soil Ingestion Sum of age groups CANCER RISK
Mirex	1.90E-07	1.10E-07	1.98E-07	6.41E-07	1.14E-06
Photomirex	5.57E-09	3.21E-09	5.81E-09	1.88E-08	3.33E-08
Sum of all chemicals					1.17E-06

FLOOD PLAIN RESIDENT: CURRENT & FUTURE USE

CHEMICAL	Soil, Dermal Adult CANCER RISK	Soil, Dermal Fifteen year old CANCER RISK	Soil, Dermal Nine year old CANCER RISK	Soil, Dermal Four year old CANCER RISK	Soil, Dermal Sum of age groups CANCER RISK
Mirex	1.32E-08	1.29E-08	1.58E-08	1.66E-08	5.85E-08
Photomirex	3.86E-10	3.79E-10	4.61E-10	4.86E-10	1.71E-09
Sum of all chemicals					6.02E-08

ENVIRON

FLOOD PLAIN RESIDENT: CURRENT & FUTURE USE

CHEMICAL	Vegetable Ingest. Adult CANCER RISK	Vegetable Ingest. Fifteen year old CANCER RISK	Vegetable Ingest. Nine year old CANCER RISK	Vegetable Ingest. Four year old CANCER RISK	Vegetable Ingest. Sum of age groups CANCER RISK	CUMULATIVE CANCER RISK
Mirex	4.39E-06	2.06E-06	3.17E-06	3.43E-06	1.31E-05	1.42E-05
Photomirex	1.40E-07	6.55E-08	1.01E-07	1.09E-07	4.15E-07	4.50E-07
Sum of all chemicals					1.35E-05	1.47E-05

FLOOD PLAIN RESIDENT: CURRENT & FUTURE USE

CHEMICAL	Soil Ingestion Adult HAZARD INDEX	Soil Ingestion Fifteen year old HAZARD INDEX	Soil Ingestion Nine year old HAZARD INDEX	Soil Ingestion Four year old HAZARD INDEX
Mirex	3.05E-01	3.81E-01	6.89E-01	2.67E+00
Photomirex	8.93E-03	1.12E-02	2.02E-02	7.81E-02
Sum of all chemicals	3.14E-01	3.92E-01	7.09E-01	2.75E+00

FLOOD PLAIN RESIDENT: CURRENT & FUTURE USE

CHEMICAL	Soil, Dermal Adult HAZARD INDEX	Soil, Dermal Fifteen year old HAZARD INDEX	Soil, Dermal Nine year old HAZARD INDEX	Soil, Dermal Four year old HAZARD INDEX
Mirex	2.12E-02	4.49E-02	5.47E-02	6.92E-02
Photomirex	6.19E-04	1.32E-03	1.60E-03	2.03E-03
Sum of all chemicals	2.18E-02	4.63E-02	5.63E-02	7.12E-02

FLOOD PLAIN RESIDENT: CURRENT & FUTURE USE

CHEMICAL	Vegetable Ingest. Adult HAZARD INDEX	Vegetable Ingest. Fifteen year old HAZARD INDEX	Vegetable Ingest. Nine year old HAZARD INDEX	Vegetable Ingest. Four year old HAZARD INDEX
Mirex	7.04E+00	7.15E+00	1.10E+01	1.43E+01
Photomirex	2.24E-01	2.28E-01	3.50E-01	4.55E-01
Sum of all chemicals	7.26E+00	7.38E+00	1.13E+01	1.48E+01

FLOOD PLAIN RESIDENT: CURRENT & FUTURE USE

Non carc Risk ?

CHEMICAL	CUMULATIVE ADULT HAZARD INDEX	CUMULATIVE FIFTEEN YEAR OLD HAZARD INDEX	CUMULATIVE NINE YEAR OLD HAZARD INDEX	CUMULATIVE FOUR YEAR OLD HAZARD INDEX
Mirex	7.37E+00	7.58E+00	1.17E+01	1.70E+01
Photomirex	2.34E-01	2.40E-01	3.72E-01	5.35E-01
Sum of all chemicals	7.60E+00	7.82E+00	1.21E+01	1.76E+01

AGRICULTURAL POPULATION

AGRICULTURAL POPULATION: FUTURE USE ONLY

CHEMICAL	Beef Ingestion Adult CANCER RISK	Beef Ingestion Fifteen year old CANCER RISK	Beef Ingestion Nine year old CANCER RISK	Beef Ingestion Four year old CANCER RISK	Beef Ingestion Sum of age groups CANCER RISK
Mirex	1.34E-05	7.33E-06	1.03E-05	1.10E-05	4.20E-05
Photomirex	4.01E-07	2.20E-07	3.09E-07	3.31E-07	1.26E-06
Sum of all chemicals					4.33E-05

AGRICULTURAL POPULATION: FUTURE USE ONLY

CHEMICAL	Milk Ingestion Adult CANCER RISK	Milk Ingestion Fifteen year old CANCER RISK	Milk Ingestion Nine year old CANCER RISK	Milk Ingestion Four year old CANCER RISK	Milk Ingestion Sum of age groups CANCER RISK	CUMULATIVE CANCER RISK
Mirex	6.93E-06	6.81E-06	1.25E-05	1.95E-05	4.57E-05	8.70E-05
Photomirex	2.04E-07	2.00E-07	3.69E-07	5.73E-07	1.35E-06	2.61E-06
Sum of all chemicals					4.71E-05	9.04E-05

AGRICULTURAL POPULATION: FUTURE USE ONLY

CHEMICAL	Beef Ingestion Adult HAZARD INDEX	Beef Ingestion Fifteen year old HAZARD INDEX	Beef Ingestion Nine year old HAZARD INDEX	Beef Ingestion Four year old HAZARD INDEX
Mirex	2.14E+01	2.54E+01	3.58E+01	4.59E+01
Photomirex	6.43E-01	7.63E-01	1.07E+00	1.38E+00
Sum of all chemicals	2.21E+01	2.62E+01	3.69E+01	4.73E+01

AGRICULTURAL POPULATION: FUTURE USE ONLY

CHEMICAL	Milk Ingestion Adult HAZARD INDEX	Milk Ingestion Fifteen year old HAZARD INDEX	Milk Ingestion Nine year old HAZARD INDEX	Milk Ingestion Four year old HAZARD INDEX
Mirex	1.11E+01	2.36E+01	4.35E+01	8.11E+01
Photomirex	3.27E-01	6.95E-01	1.28E+00	2.39E+00
Sum of all chemicals	1.14E+01	2.43E+01	4.48E+01	8.35E+01

AGRICULTURAL POPULATION: FUTURE USE ONLY

CHEMICAL	CUMULATIVE ADULT HAZARD INDEX	CUMULATIVE FIFTEEN YEAR OLD HAZARD INDEX	CUMULATIVE NINE YEAR OLD HAZARD INDEX	CUMULATIVE FOUR YEAR OLD HAZARD INDEX
Mirex	3.25E+01	4.91E+01	7.93E+01	1.27E+02
Photomirex	9.70E-01	1.46E+00	2.35E+00	3.76E+00
Sum of all chemicals	3.35E+01	5.05E+01	8.17E+01	1.31E+02

RECREATIONAL POPULATION:
UPSTREAM - CURRENT USE

RECREATIONAL SCENARIO: UPSTREAM - CURRENT USE

CHEMICAL	Sediment Ingestion Adult CANCER RISK	Sediment Ingestion Fifteen year old CANCER RISK	Sediment Ingestion Nine year old CANCER RISK	Sediment Ingestion Four year old CANCER RISK	Sediment Ingestion Sum of age groups CANCER RISK
Mirex	6.84E-09	7.44E-09	1.34E-08	2.30E-08	5.07E-08
Photomirex	1.58E-10	1.72E-10	3.10E-10	5.31E-10	1.17E-09
Sum of all chemicals					5.19E-08

RECREATIONAL SCENARIO: UPSTREAM - CURRENT USE

CHEMICAL	Sediment, Dermal Adult CANCER RISK	Sediment, Dermal Fifteen year old CANCER RISK	Sediment, Dermal Nine year old CANCER RISK	Sediment, Dermal Four year old CANCER RISK	Sediment, Dermal Sum of age groups CANCER RISK
Mirex	4.95E-09	4.89E-09	5.95E-09	3.33E-09	1.91E-08
Photomirex	1.14E-10	1.13E-10	1.37E-10	7.67E-11	4.41E-10
Sum of all chemicals					1.95E-08

RECREATIONAL SCENARIO: UPSTREAM - CURRENT USE

CHEMICAL	Fish Ingestion Adult CANCER RISK	Fish Ingestion Fifteen year old CANCER RISK	Fish Ingestion Nine year old CANCER RISK	Fish Ingestion Four year old CANCER RISK	Fish Ingestion Sum of age groups CANCER RISK	CUMULATIVE CANCER RISK
Mirex	3.78E-06	1.53E-06	2.42E-06	3.01E-06	1.07E-05	1.08E-05
Photomirex	8.79E-08	3.55E-08	5.62E-08	7.00E-08	2.50E-07	2.51E-07
Sum of all chemicals					1.10E-05	1.11E-05

RECREATIONAL SCENARIO: UPSTREAM - CURRENT USE

CHEMICAL	Sediment Ingestion Adult HAZARD INDEX	Sediment Ingestion Fifteen year old HAZARD INDEX	Sediment Ingestion Nine year old HAZARD INDEX	Sediment Ingestion Four year old HAZARD INDEX
Mirex	1.10E-02	2.58E-02	4.67E-02	9.59E-02
Photomirex	2.53E-04	5.96E-04	1.08E-03	2.21E-03
Sum of all chemicals	1.12E-02	2.64E-02	4.77E-02	9.81E-02

RECREATIONAL SCENARIO: UPSTREAM - CURRENT USE

CHEMICAL	Sediment, Dermal Adult HAZARD INDEX	Sediment, Dermal Fifteen year old HAZARD INDEX	Sediment, Dermal Nine year old HAZARD INDEX	Sediment, Dermal Four year old HAZARD INDEX
Mirex	7.93E-03	1.70E-02	2.06E-02	1.39E-02
Photomirex	1.83E-04	3.91E-04	4.76E-04	3.20E-04
Sum of all chemicals	8.11E-03	1.74E-02	2.11E-02	1.42E-02

ENVIRON

need total Sediment Intake HI!

RECREATIONAL SCENARIO: UPSTREAM - CURRENT USE

CHEMICAL	Fish Ingestion Adult HAZARD INDEX	Fish Ingestion Fifteen year old HAZARD INDEX	Fish Ingestion Nine year old HAZARD INDEX	Fish Ingestion Four year old HAZARD INDEX
Mirex	6.06E+00	5.30E+00	8.39E+00	1.25E+01
Photomirex	1.41E-01	1.23E-01	1.95E-01	2.92E-01
Sum of all chemicals	6.20E+00	5.42E+00	8.59E+00	1.28E+01

RECREATIONAL SCENARIO: UPSTREAM - CURRENT USE

CHEMICAL	CUMULATIVE ADULT HAZARD INDEX	CUMULATIVE FIFTEEN YEAR OLD HAZARD INDEX	CUMULATIVE NINE YEAR OLD HAZARD INDEX	CUMULATIVE FOUR YEAR OLD HAZARD INDEX
Mirex	6.08E+00	5.34E+00	8.46E+00	1.27E+01
Photomirex	1.41E-01	1.24E-01	1.97E-01	2.94E-01
Sum of all chemicals	6.22E+00	5.47E+00	8.65E+00	1.30E+01

RECREATIONAL POPULATION:
UPSTREAM - FUTURE USE

RECREATIONAL SCENARIO: UPSTREAM - FUTURE USE

CHEMICAL	Sediment Ingestion Adult CANCER RISK	Sediment Ingestion Fifteen year old CANCER RISK	Sediment Ingestion Nine year old CANCER RISK	Sediment Ingestion Four year old CANCER RISK	Sediment Ingestion Sum of age groups CANCER RISK
Mirex	1.37E-08	1.48E-08	2.67E-08	4.60E-08	1.01E-07
Photomirex	3.15E-10	3.41E-10	6.15E-10	1.06E-09	2.33E-09
Sum of all chemicals					1.03E-07

RECREATIONAL SCENARIO: UPSTREAM - FUTURE USE

CHEMICAL	Sediment, Dermal Adult CANCER RISK	Sediment, Dermal Fifteen year old CANCER RISK	Sediment, Dermal Nine year old CANCER RISK	Sediment, Dermal Four year old CANCER RISK	Sediment, Dermal Sum of age groups CANCER RISK
Mirex	9.89E-09	9.70E-09	1.18E-08	6.65E-09	3.81E-08
Photomirex	2.28E-10	2.24E-10	2.72E-10	1.53E-10	8.78E-10
Sum of all chemicals					3.89E-08

ENVIRON

RECREATIONAL SCENARIO: UPSTREAM - FUTURE USE

CHEMICAL	Fish Ingestion Adult CANCER RISK	Fish Ingestion Fifteen year old CANCER RISK	Fish Ingestion Nine year old CANCER RISK	Fish Ingestion Four year old CANCER RISK	Fish Ingestion Sum of age groups CANCER RISK	CUMULATIVE CANCER RISK
Mirex	4.91E-05	1.98E-05	3.14E-05	3.91E-05	1.40E-04	1.40E-04
Photomirex	1.14E-06	4.62E-07	7.31E-07	9.10E-07	3.25E-06	3.25E-06
Sum of all chemicals					1.43E-04	1.43E-04

RECREATIONAL SCENARIO: UPSTREAM - FUTURE USE

CHEMICAL	Sediment Ingestion Adult HAZARD INDEX	Sediment Ingestion Fifteen year old HAZARD INDEX	Sediment Ingestion Nine year old HAZARD INDEX	Sediment Ingestion Four year old HAZARD INDEX
Mirex	2.19E-02	5.13E-02	9.26E-02	1.92E-01
Photomirex	5.05E-04	1.18E-03	2.14E-03	4.42E-03
Sum of all chemicals	2.24E-02	5.25E-02	9.48E-02	1.96E-01

RECREATIONAL SCENARIO: UPSTREAM - FUTURE USE

CHEMICAL	Sediment, Dermal Adult HAZARD INDEX	Sediment, Dermal Fifteen year old HAZARD INDEX	Sediment, Dermal Nine year old HAZARD INDEX	Sediment, Dermal Four year old HAZARD INDEX
Mirex	1.59E-02	3.37E-02	4.10E-02	2.77E-02
Photomirex	3.66E-04	7.77E-04	9.45E-04	6.39E-04
Sum of all chemicals	1.62E-02	3.45E-02	4.19E-02	2.84E-02

RECREATIONAL SCENARIO: UPSTREAM - FUTURE USE

CHEMICAL	Fish Ingestion Adult HAZARD INDEX	Fish Ingestion Fifteen year old HAZARD INDEX	Fish Ingestion Nine year old HAZARD INDEX	Fish Ingestion Four year old HAZARD INDEX
Mirex	7.88E+01	6.89E+01	1.09E+02	1.63E+02
Photomirex	1.83E+00	1.60E+00	2.54E+00	3.79E+00
Sum of all chemicals	8.06E+01	7.05E+01	1.12E+02	1.67E+02

RECREATIONAL SCENARIO: UPSTREAM - FUTURE USE

CHEMICAL	CUMULATIVE ADULT HAZARD INDEX	CUMULATIVE FIFTEEN YEAR OLD HAZARD INDEX	CUMULATIVE NINE YEAR OLD HAZARD INDEX	CUMULATIVE FOUR YEAR OLD HAZARD INDEX
Mirex	7.88E+01	6.90E+01	1.09E+02	1.63E+02
Photomirex	1.83E+00	1.60E+00	2.54E+00	3.80E+00
Sum of all chemicals	8.06E+01	7.06E+01	1.12E+02	1.67E+02

RECREATIONAL POPULATION:
DOWNSTREAM - CURRENT AND FUTURE USE

RECREATIONAL SCENARIO: DOWNSTREAM - CURRENT & FUTURE USE

CHEMICAL	Sediment Ingestion Adult CANCER RISK	Sediment Ingestion Fifteen year old CANCER RISK	Sediment Ingestion Nine year old CANCER RISK	Sediment Ingestion Four year old CANCER RISK	Sediment Ingestion Sum of age groups CANCER RISK
Mirex	4.66E-10	5.03E-10	9.09E-10	1.57E-09	3.45E-09
Photomirex	NC	NC	NC	NC	NA
Sum of all chemicals					3.45E-09

RECREATIONAL SCENARIO: DOWNSTREAM - CURRENT & FUTURE USE

CHEMICAL	Sediment, Dermal Adult CANCER RISK	Sediment, Dermal Fifteen year old CANCER RISK	Sediment, Dermal Nine year old CANCER RISK	Sediment, Dermal Four year old CANCER RISK	Sediment, Dermal Sum of age groups CANCER RISK
Mirex	3.37E-10	3.30E-10	4.02E-10	2.27E-10	1.30E-09
Photomirex	NC	NC	NC	NC	NA
Sum of all chemicals					1.30E-09

RECREATIONAL SCENARIO: DOWNSTREAM - CURRENT & FUTURE USE

CHEMICAL	Fish Ingestion Adult CANCER RISK	Fish Ingestion Fifteen year old CANCER RISK	Fish Ingestion Nine year old CANCER RISK	Fish Ingestion Four year old CANCER RISK	Fish Ingestion Sum of age groups CANCER RISK	CUMULATIVE CANCER RISK
Mirex	3.14E-06	1.27E-06	2.01E-06	2.50E-06	8.93E-06	8.93E-06
Photomirex	2.97E-07	1.20E-07	1.90E-07	2.37E-07	8.44E-07	8.44E-07
Sum of all chemicals					9.77E-06	9.77E-06

RECREATIONAL SCENARIO: DOWNSTREAM - CURRENT & FUTURE USE

CHEMICAL	Sediment Ingestion Adult HAZARD INDEX	Sediment Ingestion Fifteen year old HAZARD INDEX	Sediment Ingestion Nine year old HAZARD INDEX	Sediment Ingestion Four year old HAZARD INDEX
Mirex	7.47E-04	1.75E-03	3.15E-03	6.53E-03
Photomirex	NC	NC	NC	NC
Sum of all chemicals	7.47E-04	1.75E-03	3.15E-03	6.53E-03

RECREATIONAL SCENARIO: DOWNSTREAM - CURRENT & FUTURE USE

CHEMICAL	Sediment, Dermal Adult HAZARD INDEX	Sediment, Dermal Fifteen year old HAZARD INDEX	Sediment, Dermal Nine year old HAZARD INDEX	Sediment, Dermal Four year old HAZARD INDEX
Mirex	5.40E-04	1.15E-03	1.40E-03	9.44E-04
Photomirex	NC	NC	NC	NC
Sum of all chemicals	5.40E-04	1.15E-03	1.40E-03	9.44E-04

RECREATIONAL SCENARIO: DOWNSTREAM - CURRENT & FUTURE USE

CHEMICAL	Fish Ingestion Adult HAZARD INDEX	Fish Ingestion Fifteen year old HAZARD INDEX	Fish Ingestion Nine year old HAZARD INDEX	Fish Ingestion Four year old HAZARD INDEX
Mirex	5.04E+00	4.41E+00	6.98E+00	1.04E+01
Photomirex	4.76E-01	4.17E-01	6.60E-01	9.86E-01
Sum of all chemicals	5.51E+00	4.82E+00	7.64E+00	1.14E+01

RECREATIONAL SCENARIO: DOWNSTREAM - CURRENT & FUTURE USE

CHEMICAL	CUMULATIVE ADULT HAZARD INDEX	CUMULATIVE FIFTEEN YEAR OLD HAZARD INDEX	CUMULATIVE NINE YEAR OLD HAZARD INDEX	CUMULATIVE FOUR YEAR OLD HAZARD INDEX
Mirex	5.04E+00	4.41E+00	6.98E+00	1.04E+01
Photomirex	4.76E-01	4.17E-01	6.60E-01	9.86E-01
Sum of all chemicals	5.51E+00	4.83E+00	7.64E+00	1.14E+01

RECREATIONAL POPULATION: GAME

GAME SCENARIO: CURRENT & FUTURE USE

CHEMICAL	Game Ingestion Adult CANCER RISK	Game Ingestion Fifteen year old CANCER RISK	Game Ingestion Nine year old CANCER RISK	Game Ingestion Four year old CANCER RISK	Game Ingestion Sum of CANCER RISK
Mirex	6.59E-08	2.66E-08	4.22E-08	5.25E-08	1.87E-07
Photomirex	NC	NC	NC	NC	NA
Sum of all chemicals					1.87E-07

GAME SCENARIO: CURRENT & FUTURE USE

CHEMICAL	Game Ingestion Adult HAZARD INDEX	Game Ingestion Fifteen year old HAZARD INDEX	Game Ingestion Nine year old HAZARD INDEX	Game Ingestion Four year old HAZARD INDEX
Mirex	1.06E-01	9.25E-02	1.46E-01	2.19E-01
Photomirex	NC	NC	NC	NC
Sum of all chemicals	1.06E-01	9.25E-02	1.46E-01	2.19E-01

TABLE 19
Intake Assumptions for Ingestion of Soil

Equation:

$$Intake\ (mg/kg/day) = \frac{CS \times IR \times 1\ kg/10^6\ mg \times FI \times BF \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CS= Chemical Concentration in Soil (mg/kg)	Estimated using site investigation data			
IR= Ingestion Rate (mg/day)[a]	100	100	100	200
FI= Fraction Ingested from Contaminated Source (unitless)[a]	0.5	0.5	0.5	0.5
BF= Bioavailability Factor (unitless)[a]	1	1	1	1
EF= Exposure Frequency (days/yr)[a]	365	365	365	365
ED= Exposure Duration (yrs)[b]	13	6	6	5
Lifetime (yrs)[c]	75	75	75	75
BW= Body Weight (kg)[a]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]See Appendix D.
[b]See text, Chapter VII.
[c]USEPA 1990c.

TABLE 20
Intake Assumptions for Ingestion of Sediment (Current Use Upstream)

Equation:

$$Intake\ (mg/kg/day) = \frac{CS \times IR \times 1\ kg/10^6\ mg \times FI \times BF \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CS= Chemical Concentration in Sediment (mg/kg)	Estimated using site investigation data .			
IR= Ingestion Rate (mg/day)[a]	100	100	100	200
FI= Fraction Ingested from Contaminated Source (unitless)[a]	0.5	0.5	0.5	0.5
BF= Bioavailability Factor (unitless)[a]	1	1	1	1
EF= Exposure Frequency (days/yr)[a]	35	66	66	35
ED= Exposure Duration (yrs)[b]	13	6	6	5
Lifetime (yrs)[c]	75	75	75	75
BW= Body Weight (kg)[c]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]See Appendix D.
[b]See text, Chapter VII.
[c]USEPA 1990c.

TABLE 21
Intake Assumptions for Ingestion of Sediment
(Current Use Downstream; Future Use Upstream and Downstream)

Equation:

$$Intake\ (mg/kg/day) = \frac{CS \times IR \times 1\ kg/10^6\ mg \times FI \times BF \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CS= Chemical Concentration in Sediment (mg/kg)	Estimated using site investigation data			
IR= Ingestion Rate (mg/day)[a]	100	100	100	200
FI= Fraction Ingested from Contaminated Source (unitless)[a]	0.5	0.5	0.5	0.5
BF= Bioavailability Factor (unitless)[a]	1	1	1	1
EF= Exposure Frequency (days/yr)[a]	70	131	131	70
ED= Exposure Duration (yrs)[b]	13	6	6	5
Lifetime (yrs)[c]	75	75	75	75
BW= Body Weight (kg)[c]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]See Appendix D.
[b]See text, Chapter VII.
[c]USEPA 1990c.

C. Dermal Contact with Soil and Sediment

Dermal exposure to the chemicals of potential concern in soil may occur when the skin of the adult or child flood plain resident comes in contact with the soil around their residence. Exposure may also occur as a result of dermal contact with sediments for the recreational population. The following factors must be taken into account when evaluating this potential route of exposure: 1) the chemical concentration in soil or sediment; 2) the skin surface area exposed to the soil or sediment; 3) the amount of soil or sediment deposited on the skin; 4) the frequency and duration of exposure; and 5) the extent to which chemicals adsorbed to the soil or sediment are subsequently absorbed through the skin.

Exposure of flood plain residents to known or suspected carcinogenic and noncarcinogenic chemicals of potential concern through dermal contact with soil were calculated using the equation and assumptions in Table 22.

As a result of the advisory posted along the MFLBC, the frequency of visits to the MFLBC in the area of the advisory and downstream of the advisory (i.e., upstream and downstream of Lisbon Dam) is likely to differ. The frequency of visits within the advisory area may also differ in the future. Current exposure to known or suspected carcinogenic and noncarcinogenic chemicals of potential concern via dermal contact with contaminated sediment upstream of Lisbon Dam (i.e., within the area of the advisory) were calculated using the equation and assumptions in Table 23. Potential exposures resulting from dermal contact with sediment currently in the downstream section of the creek and in the future in both upstream and downstream sections of the creek were calculated using the assumptions in Table 24.

D. Ingestion of Homegrown Beef and Milk

Ingestion of contaminated sediment, soil, and grass by livestock and the bioaccumulation of certain chemicals in meat and milk could pose a potential pathway of exposure for local farm families that consume milk and beef from cattle raised on their farms. Because the creek and portions of the flood plain have been fenced off on property owned by dairy farmers, this pathway does not present a current source of exposure. Potential exposures are characterized under the future exposure scenario because, as noted previously, USEPA guidance (USEPA 1989a) requires that exposures be calculated as if these remedial measures were not put in place.

TABLE 22
Intake Assumptions for Dermal Contact With Soil

Equation:

$$Intake\ (mg/kg/day) = \frac{CS \times 1\ kg/10^6\ mg \times FC \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CS= Chemical Concentration in Soil (mg/kg)	Estimated using site investigation data			
FC= Fraction Contacted from Contaminated Source	0.5	0.5	0.5	0.5
SA= Skin Surface Area Available for Contact (cm^2/day)[a]	4019	3650	2459	1606
AF= Soil to Skin Adherence Factor (mg/cm^2)[a]	0.9	0.9	0.9	0.9
ABS= Absorption Factor (unitless)[b]	0.01	0.01	0.01	0.01
EF= Exposure Frequency (days/yr)[a]	70	131	131	131
ED= Exposure Duration (yrs)[c]	13	6	6	5
Lifetime (yrs)[d]	75	75	75	75
BW= Body Weight (kg)[d]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	4745 27375	2190 27375	2190 27375	1825 27375

[a]See Appendix D.
[b]See Chapter IV.
[c]See text, Chapter VII.
[d]USEPA 1990c.

TABLE 23
Intake Assumptions for Dermal Contact With Sediment (Current Use Upstream)

Equation:

$$Intake\ (mg/kg/day) = \frac{CS \times 1\ kg/10^6\ mg \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CS= Chemical Concentration in Sediment (mg/kg)	Estimated using site investigation data			
SA= Skin Surface Area Available for Contact (cm^2/day)[a]	4019	3650	2459	1606
AF= Sediment to Skin Adherence Factor (mg/cm^2)[a]	0.9	0.9	0.9	0.9
ABS= Absorption Factor (unitless)[b]	0.01	0.01	0.01	0.01
EF= Exposure Frequency (days/yr)[a]	35	66	66	35
ED= Exposure Duration (yrs)[c]	13	6	6	5
Lifetime (yrs)[d]	75	75	75	75
BW= Body Weight (kg)[d]	70	56	31	16
AT= Averaging Time (days)				
Noncarcinogens	4745	2190	2190	1825
Carcinogens	27375	27375	27375	27375

[a]See Appendix D.
[b]See Chapter IV.
[c]See text, Chapter VII.
[d]USEPA 1990c.

TABLE 24
Intake Assumptions for Dermal Contact With Sediment
(Current Use Downstream; Future Use Upstream and Downstream)

Equation:

$$Intake\ (mg/kg/day) = \frac{CS \times 1\ kg/10^6\ mg \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CS= Chemical Concentration in Sediment (mg/kg)	Estimated using site investigation data .			
SA= Skin Surface Area Available for Contact (cm^2/day)[a]	4019	3650	2459	1606
AF= Sediment to Skin Adherence Factor (mg/cm^2)[a]	0.9	0.9	0.9	0.9
ABS= Absorption Factor (unitless)[b]	0.01	0.01	0.01	0.01
EF= Exposure Frequency (days/yr)[a]	70	131	131	70
ED= Exposure Duration (yrs)[a]	13	6	6	5
Lifetime (yrs)[d]	75	75	75	75
BW= Body Weight (kg)[d]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]See Appendix D.
[b]See Chapter IV.
[c]See text, Chapter VII.
[d]USEPA 1990c.

The following factors must be taken into account when evaluating this potential route of exposure: 1) the chemical concentration in food (milk or beef); 2) the rate of ingestion; 3) the fraction of total milk or beef ingested that is homegrown; and 4) the frequency and duration of exposure.

Exposures to known or suspected carcinogenic and noncarcinogenic chemicals of potential concern via beef ingestion were calculated using the equation and assumptions in Table 25. The equation and values used to calculate exposure from the consumption of contaminated milk are provided in Table 26.

E. Ingestion of Homegrown Vegetables

Flood plain residents could potentially be exposed to the chemicals of potential concern through ingestion of vegetables grown in flood plain soil. The following factors must be taken into consideration when estimating this exposure: 1) the chemical concentration in vegetables; 2) the rate of consumption of vegetables; 3) the fraction of total vegetables that is homegrown; and 4) the frequency and duration of exposure.

Exposures to known or suspected carcinogenic and noncarcinogenic chemicals of potential concern via ingestion of homegrown vegetables were calculated using the equation and assumptions in Table 27.

F. Ingestion of Fish

Local residents may be exposed to the chemicals of potential concern through the ingestion of fish caught by recreational fishermen from the MFLBC. The following factors must be taken into consideration when estimating this exposure: 1) the chemical concentration in fish; 2) the rate of fish ingestion; 3) the fraction of fish consumed that were caught in an affected area; and 4) the frequency and duration of exposure.

As a result of the advisory posted along the MFLBC, the amount of fishing in the area downstream of the advisory (i.e., downstream of Lisbon Dam) is likely to be higher than that within the advisory area. A future exposure scenario for the area within the advisory was also developed because USEPA guidance (USEPA 1989a) requires that hypothetical exposures be assessed in the absence of any actions to control or mitigate site-related releases (i.e., as if the advisory were not in place).

TABLE 25
Intake Assumptions for Ingestion of Beef

Equation:

$$Intake\ (mg/kg/day) = \frac{CF \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CF= Chemical Concentration in Food (beef) (mg/kg)	Estimated using site investigation data			
IR= Ingestion Rate (kg/day)[a]	0.100	0.095	0.074	0.049
FI= Fraction Ingested from Contaminated Source (unitless)[b]	0.75	0.75	0.75	0.75
EF= Exposure Frequency (days/yr)[b]	365	365	365	365
ED= Exposure Duration (yrs)[c]	13	6	6	5
Lifetime (yrs)[d]	75	75	75	75
BW= Body Weight (kg)[d]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]USEPA 1990c for adults; see Appendix D for rates for children.
[b]See Appendix D.
[c]See text, Chapter VII.
[d]USEPA 1990c.

TABLE 26
Intake Assumptions for Ingestion of Milk

Equation:

$$Intake\ (mg/kg/day) = \frac{CF \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CF= Chemical Concentration in Food (Milk) (mg/kg)	Estimated using site investigation data			
IR= Ingestion Rate (kg/day)[a]	0.305	0.519	0.529	0.509
FI= Fraction Ingested from Contaminated Source (unitless)[b]	0.75	0.75	0.75	0.75
EF= Exposure Frequency (days/yr)[b]	365	365	365	365
ED= Exposure Duration (yrs)[c]	13	6	6	5
Lifetime (yrs)[d]	75	75	75	75
BW= Body Weight (kg)[d]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]USEPA 1990c for adults; see Appendix D for rates for children.
[b]See Appendix D.
[c]See text, Chapter VII.
[d]USEPA 1990c.

TABLE 27
Intake Assumptions for Ingestion of Homegrown Vegetables

Equation:

$$Intake\ (mg/kg/day) = \frac{CF \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CF= Chemical Concentration in Food (vegetables) (mg/kg)	Estimated using site investigation data			
IR= Ingestion Rate (kg/day)[a]	0.224	0.182	0.155	0.104
FI= Fraction Ingested from Contaminated Source (unitless)[a]	0.40	0.40	0.40	0.40
EF= Exposure Frequency (days/yr)[a]	183	183	183	183
ED= Exposure Duration (yrs)[b]	13	6	6	5
Lifetime (yrs)[c]	75	75	75	75
BW= Body Weight (kg)[c]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]See Appendix D.
[b]See text, Chapter VII.
[c]USEPA 1990c.

Current exposure to known or suspected carcinogenic and noncarcinogenic chemicals of potential concern via ingestion of fish caught upstream of Lisbon Dam (i.e., in the area of the advisory) were calculated using the equation and assumptions in Table 28. Potential exposures resulting from ingestion of fish caught currently downstream and in the future in both upstream and downstream sections of the creek were calculated using the assumptions in Table 29.

G. Ingestion of Game

Local residents could potentially be exposed to the chemicals of potential concern through the ingestion of game (e.g., opossum, raccoon, squirrel and deer) killed by hunters in the area surrounding the MFLBC. The following factors must be taken into consideration when estimating this exposure: 1) the chemical concentration in the game; 2) the rate of ingestion of game; 3) the fraction of game consumed that is contaminated; and 4) the exposure frequency and duration.

Exposures to known or suspected carcinogenic and noncarcinogenic chemicals of potential concern via ingestion of hunted game were calculated using the equation and assumptions in Table 30.

As previously noted, the ODH concluded that consumption of raccoons and opossums hunted or trapped in the MFLBC watershed should not pose a significant risk to human health. (See Section VI.G. for additional discussion of ODH conclusions regarding the residue levels of mirex in wildlife.)

TABLE 28
Intake Assumptions for Ingestion of Fish
(Current Use Upstream)

Equation:

$$Intake\ (mg/kg/day) = \frac{CF \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CF= Chemical Concentration in Fish (mg/kg)	Estimated using site investigation data			
IR= Ingestion Rate (kg/day)[a]	0.150	0.105	0.092	0.071
FI= Fraction Ingested from Contaminated Source (unitless)[a]	1	1	1	1
EF= Exposure Frequency (days/yr)[a]	4	4	4	4
ED= Exposure Duration (yrs)[b]	13	6	6	5
Lifetime (yrs)[a]	75	75	75	75
BW= Body Weight (kg)[a]	70	56	31	16
AT= Averaging Time (days)				
Noncarcinogens	4745	2190	2190	1825
Carcinogens	27375	27375	27375	27375

[a]See Appendix D.
[b]See text, Chapter VII.
[c]USEPA 1990c.

TABLE 29
Intake Assumptions for Ingestion of Fish
(Current Use Downstream; Future Use Upstream and Downstream)

Equation:

$$Intake\ (mg/kg/day) = \frac{CF \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CF= Chemical Concentration in Fish (mg/kg)	Estimated using site investigation data			
IR= Ingestion Rate (kg/day)[a]	0.150	0.105	0.092	0.071
FI= Fraction Ingested from Contaminated Source (unitless)[a]	1	1	1	1
EF= Exposure Frequency (days/yr)[a]	52	52	52	52
ED= Exposure Duration (yrs)[b]	13	6	6	5
Lifetime (yrs)[c]	75	75	75	75
BW= Body Weight (kg)[c]	70	56	31	16
AT= Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]See Appendix D.
[b]See text, Chapter VII.
[c]USEPA 1990c.

low IRs

TABLE 30
Intake Assumptions for Ingestion of Game

Equation:

$$Intake\ (mg/kg/day) = \frac{CF \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter Case	Adult	15 year old	9 year old	4 year old
CF = Chemical Concentration in Food (Game)(mg/kg)	Estimated using site investigation data			
IR = Ingestion Rate (kg/day)[a]	0.150	0.105	0.092	0.071
FI = Fraction Ingested from Contaminated Source (unitless)[a]	1	1	1	1
EF = Exposure Frequency (days/yr)[a]	12	12	12	12
ED = Exposure Duration (yrs)[b]	13	6	6	5
Lifetime (yrs)[c]	75	75	75	75
BW = Body Weight (kg)[a]	70	56	31	16
AT = Averaging Time (days) Noncarcinogens Carcinogens	 4745 27375	 2190 27375	 2190 27375	 1825 27375

[a]See Appendix D.
[b]See text, Chapter VII.
[c]USEPA 1990c.

VIII. RISK CHARACTERIZATION

A. Introduction

Risk characterization is the final step of the public health risk assessment process, as described in Chapter I. In this step, the toxicity values (i.e., SFs and RfDs) for the chemicals of potential concern are used in conjunction with the estimated chemical intakes for the modeled populations to estimate quantitatively both carcinogenic and noncarcinogenic health risks. The methodology for deriving quantitative risk estimates is presented in Section B below. Section C of this chapter presents the baseline risk estimates for the hypothetical current use and future use scenarios addressed in this risk assessment.

As discussed in Chapter VII, each scenario was modeled for the RME condition. Based on USEPA guidance (1989a), the RME is used to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposures.

It is important for the reader to understand that the risk values estimated in this assessment are not actuarial risks, i.e., they are not risks that have been documented as a result of human exposure to the chemicals of potential concern. As discussed in Section I.B. of this baseline risk assessment, The Risk Assessment Process, risk estimates are based on a series of conservative assumptions and, as such, represent an upper bound on risk. The risk values presented below are useful because they can be compared with other risks that have been estimated using the same procedures. Perhaps the most useful application of the quantitative risk estimates that follow is as a means for identifying the most significant potential exposure pathways in terms of potential health risk.

The numerical risk estimates that are presented in this chapter must be interpreted in the context of the uncertainties and assumptions associated with each step of the risk assessment process. Some of the major uncertainties and assumptions associated with this risk assessment are discussed in Section D of the chapter.

B. Methodology for Quantitative Risk Estimation

1. Estimation of Cancer Risks

The numerical estimate of the excess lifetime cancer risk resulting from the modeled exposure to a specific carcinogenic chemical of potential concern can be calculated by multiplying the chronic daily intake (CDI) by the risk per unit dose, or

SF, as follows:

$$Risk = CDI \times SF$$

where:

Risk	=	lifetime probability of developing cancer due to exposure to the chemical of potential concern
CDI	=	chronic daily intake, mg/kg/day
SF	=	carcinogenic slope factor, $(mg/kg/day)^{-1}$

Regulatory agencies generally make the conservative assumption that any internal dose of any chemical classified as carcinogenic, no matter how small, presents some carcinogenic risk to humans. As discussed below in the section on uncertainties, however, the hypothesis that no threshold dose exists for carcinogens is by no means proven, and may not hold for some carcinogens that do not appear to act directly on genetic material (DNA). In cases of multiple chemical exposures, regulatory agencies also assume cancer risks to be additive (USEPA 1986b, 1989a). Accordingly, the risk estimates summarized in this chapter are the sums of the risk estimates for both chemicals of potential concern -- mirex and photomirex -- evaluated in this assessment.

In interpreting the significance of the cancer risk estimates, the reader should consider USEPA policy. The Agency has made it clear that it does not consider any specific cancer risk level as representing an insignificant risk. Instead, the USEPA has adopted a risk range. In the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) (40 CFR Part 300), USEPA states that: "For known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} and 10^{-6} using information on the relationship between dose and response." In the evaluation of estimated cancer risks developed in this assessment (see below), cancer risks are evaluated in light of the range of risks generally regarded as acceptable by USEPA.

2. Estimation of Risks for Noncancer Effects

Unlike the measure of risk used for carcinogens, the measure used to describe the potential for noncarcinogenic toxicity to occur is not expressed as a probability of

experiencing an adverse effect. Instead, the numerical estimate of the potential for adverse noncancer effects resulting from exposure to a chemical of potential concern is derived in the following manner:

Potential for adverse effects = CDI/RfD

where:

CDI = Chronic daily intake, mg/kg/day
RfD = Reference Dose, mg/kg/day

If the resulting ratio, also referred to as the hazard quotient, is less than or equal to one, it is assumed that the exposed population would not be affected. If the hazard quotient is greater than one, there may be concern for potential noncancer effects. A hazard quotient that is greater than one should not be interpreted to mean that adverse effects will occur because of the uncertainty (safety) factors used in estimating the RfD and the conservative assumptions used in estimating the CDI that tend to overestimate exposure. As a rule, however, the greater the value of the hazard quotient above one, the greater the level of concern.

As a first screening, the hazard quotients for individual chemicals can be added for any single pathway to estimate the occurrence and severity of toxic effects resulting from exposure to multiple contaminants. The USEPA (1989a) calls these summed quotients the Hazard Index (HI). The HI approach assumes that multiple sub-threshold (below the RfD) exposures could result in an adverse effect and that a reasonable criterion for evaluating the potential for adverse effects is the sum of the hazard quotients. If the HI is less than one, cumulative exposure to the substances of interest would probably not result in adverse effects. If the HI is greater than one, there is an increased potential for adverse effects under the assumed exposure conditions. An HI greater than one, however, does not necessarily indicate that the multiple exposure would harm individuals. According to USEPA (1986b, 1989a), this methodology is most properly applied to substances that induce the same effect on the same target organs. Consequently, application of the HI methodology to a mixture of substances that are not expected to induce the same effect on the same organs would likely overestimate the potential for adverse health effects.

C. Risk Estimates

Tables 31 and 32 summarize the lifetime excess cancer risk estimates for the chemicals of potential concern, mirex and photomirex, under the scenarios considered in the baseline risk assessment. Tables 33 and 34 summarize the hazard indices for mirex and photomirex under the scenarios considered in the baseline risk assessment. Appendix E contains tables that list estimated cancer risks and hazard quotients for each of the chemicals of potential concern for each of the modeled pathways.

1. Current Use Scenario

a. *Flood Plain Resident*

This scenario modeled exposure to a flood plain resident via dermal contact and incidental ingestion of soils while gardening or playing, and the ingestion of home grown vegetables. The cancer risk from soil ingestion is approximately 1×10^{-6}; from dermal contact with soil is approximately 6×10^{-8}; and from ingestion of vegetables is approximately 1×10^{-5}. The HI from soil ingestion is approximately 3; from dermal contact with soil is approximately 0.07; and from ingestion of vegetables is approximately 15.

b. Agricultural Population

Because Ruetgers-Nease has installed fences on dairy farms with land adjacent to the MFLBC to prevent access of livestock to the creek, there are no current exposures to local dairy farm families (the "agricultural population") via ingestion of milk or beef.

c. Recreational Population

The current use scenario modeled exposure to the recreational population during activities such as fishing, swimming, and wading. During these activities, potential exposure would be via incidental ingestion of and dermal contact with sediments, surface water and the via ingestion of fish. Because an advisory against fishing, wading, and swimming is in effect in the stretch of the MFLBC upstream of Lisbon Dam, exposures are assumed to differ within and outside the advisory area. In addition to the above pathways, the recreational population is assumed to be exposed via ingestion of game hunted

TABLE 31
Estimated Cancer Risks Associated With the Middle Fork of Little Beaver Creek Current Use[1]

Exposure Medium/ Exposure Route	Flood Plain Resident	Agricultural Population	Recreational Population
Soil			
Incidental ingestion	1×10^{-6}		
Dermal contact	6×10^{-8}		
Sediment			
Incidental ingestion			$5 \times 10^{-8}/3 \times 10^{-9}$ (2)
Dermal contact			$2 \times 10^{-8}/1 \times 10^{-9}$ (2)
Food			
Ingestion of vegetables	1×10^{-5}		
Ingestion of beef			
Ingestion of milk			
Ingestion of fish			$1 \times 10^{-5}/1 \times 10^{-5}$ (2)
Ingestion of game			2×10^{-7}

[1] Note: The cancer risk values presented in this table are developed using risk assessment methods described in Chapter I (Section B) and Chapter VIII. These values are upper bound risk estimates and do not represent actuarial risks.

[2] Upstream of Lisbon Dam/Downstream of Lisbon Dam

TABLE 32
Estimated Cancer Risks Associated With the Middle Fork of Little Beaver Creek Future Use[1]

Exposure Medium/ Exposure Route	Flood Plain Resident	Agricultural Population	Recreational Population
Soil			
Incidental ingestion	1×10^{-6}		
Dermal contact	6×10^{-8}		
Sediment			
Incidental ingestion			$1 \times 10^{-7}/3 \times 10^{-9}$ (2)
Dermal contact			$4 \times 10^{-8}/1 \times 10^{-9}$ (2)
Food			
Ingestion of vegetables	1×10^{-5}		
Ingestion of beef		4×10^{-5}	
Ingestion of milk		5×10^{-5}	
Ingestion of fish			$1 \times 10^{-4}/1 \times 10^{-5}$ (2)
Ingestion of game			2×10^{-7}

[1] Note: The cancer risk values presented in this table are developed using risk assessment methods described in Chapter I (Section B) and Chapter VIII. These values are upper bound risk estimates and do not represent actuarial risks.

[2]Upstream of Lisbon Dam/Downstream of Lisbon Dam

TABLE 33
Noncancer Hazard Index Estimates Associated With the Middle Fork of Little Beaver Creek Current Use[1,2]

Exposure Medium/ Exposure Route	Flood Plain Resident	Agricultural Population	Recreational Population
Soil			
Incidental ingestion	3		
Dermal contact	0.07		
Sediment			
Incidental ingestion			0.1/0.006 (3)
Dermal contact			0.02/0.001 (3)
Food			
Ingestion of vegetables	15		
Ingestion of beef			
Ingestion of milk			
Ingestion of fish			13/11 (3)
Ingestion of game			0.2

[1] Note: The hazard index (HI) values presented in this table are developed using risk assessment methods described in Chapter I (Section B) and Chapter VIII. As described in these chapters, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity. The resulting HI values do not represent actuarial risks.

[2] The values represent the noncancer hazard indices for the most sensitive age group for each route of exposure in each potentially exposed population.

[3] Upstream of Lisbon Dam/Downstream of Lisbon Dam

TABLE 34
Noncancer Hazard Index Estimates Associated With the Middle Fork of Little Beaver Creek Future Use[1, 2]

Exposure Medium/ Exposure Route	Flood Plain Resident	Agricultural Population	Recreational Population
Soil			
Incidental ingestion	3		
Dermal contact	0.07		
Sediment			
Incidental ingestion			0.2/0.006 (3)
Dermal contact			0.04/0.001 (3)
Food			
Ingestion of vegetables	15		
Ingestion of beef		47	
Ingestion of milk		84	
Ingestion of fish			167/11 (3)
Ingestion of game			0.2

[1] Note: The hazard index (HI) values presented in this table are developed using risk assessment methods described in Chapter I (Section B) and Chapter VIII. As described in these chapters, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity. The resulting HI values do not represent actuarial risks.

[2]The values represent the noncancer hazard indices for the most sensitive age group for each route of exposure in each potentially exposed population.

[3]Upstream of Lisbon Dam/Downstream of Lisbon Dam

or trapped along the MFLBC.

The cancer risks from sediment ingestion are approximately 5×10^{-8} (upstream of Lisbon Dam) and 3×10^{-9} (downstream of Lisbon Dam); from dermal contact with sediment are approximately 2×10^{-8} (upstream) and 1×10^{-9} (downstream); from ingestion of fish are approximately 1×10^{-5} (upstream) and 1×10^{-5} (downstream); and from ingestion of game is approximately 2×10^{-7}.

The HI values from sediment ingestion are approximately 0.1 (upstream of Lisbon Dam) and 0.006 (downstream of Lisbon Dam); from dermal contact with sediment are approximately 0.02 (upstream) and 0.001 (downstream); from ingestion of fish are approximately 13 (upstream) and 11 (downstream); and from ingestion of game is approximately 0.2.

2. Future Use Scenario

a. Flood Plain Resident

Exposures were assumed to be the same for the flood plain resident under the current and future use scenarios. Therefore, the risk estimates for the flood plain resident for the future use scenario are the same as those presented above for the current use scenario.

b. Agricultural Population

As required by USEPA guidance, potential exposures and associated risks for local dairy farm families (the "agricultural population") via the ingestion of beef and milk have been assessed as though the fencing that currently restricts access of local cattle to the MFLBC was not in place. The cancer risks from ingestion of beef is approximately 4×10^{-5}, and from ingestion of milk approximately 5×10^{-5}. The HI from ingestion of beef is approximately 47, and from ingestion of milk is approximately 84.

c. Recreational Population

The future use scenario assumes the same exposure pathways for the recreational population as those in the current use scenario. In the future, however, potential exposures and associated risks upstream of the Lisbon Dam

have been assessed as though the current advisory against fishing, wading, and swimming were not in place. Therefore, exposure assumptions for the upstream portion of the MFLBC under the current and future use scenarios differ; under the future use scenario, exposures are assumed to be the same as those downstream of the dam. Exposure assumptions for the portion of the MFLBC downstream of the Lisbon Dam are the same for both the current and future use scenarios, which results in the same risk estimates.

The upstream cancer risk from sediment ingestion under the future use scenario is approximately 1×10^{-7}; from dermal contact with sediment is approximately 4×10^{-8}; and from ingestion of fish is approximately 1×10^{-4}. The upstream HI from sediment ingestion is approximately 0.2; from dermal contact with sediment is approximately 0.04; and from ingestion of fish is approximately 167.

As noted above, the estimated risks from contact with the MFLBC downstream of Lisbon Dam under the future use scenario are the same as those under the current use scenario. Estimated risks from ingestion of game are also the same under both the current and future use scenarios.

3. Discussion of Risk Estimates

a. Carcinogenic Risks

USEPA has stated in the NCP that risks in the range of 10^{-4} to 10^{-6} are acceptable. In this baseline risk assessment of the MFLBC, a number of pathways pose risks less than 10^{-6} under both the current and future use scenarios, including incidental dermal contact with soil by individuals whose residences are in the flood plain, incidental ingestion of creek sediment and dermal contact with sediment by local residents who visit the creek for recreational activities, and ingestion of game by local hunters. Incidental ingestion of soil by flood plain residents poses a risk at the lower end of the acceptable risk range of 1×10^{-6}. Thus, those pathways involving direct contact with soil and sediment containing mirex and photomirex, as well as consumption of local game, pose no significant risks to local populations even under the conservative assumptions used in this risk assessment.

Under both the current and future use scenarios, several pathways

involving indirect exposure to mirex, including ingestion of vegetables by flood plain residents, ingestion of fish from the MFLBC, and ingestion of beef and milk by local farmers were evaluated. All of the estimated risk values for these pathways are within the range of risks (i.e., 10^{-4} to 10^{-6}) considered acceptable by USEPA.

b. Noncarcinogenic Risks

As noted above, the method used to describe the potential for noncarcinogenic toxicity to occur is not expressed as a probability of incurring adverse health effects as in the carcinogenic risk assessment. The HI is a ratio of the estimated exposure level to the RfD. Regulatory agencies traditionally assume that the target HI value of one or less indicates no concern for potential noncancer effects. It is important to recognize that at an HI of one, the estimated exposure is far below that exposure required to produce demonstrable toxicity in the most sensitive species tested. Thus, the estimated exposure may even exceed the RfD without a significant risk arising, although as the estimated exposure approaches the experimental NOAEL upon which the RfD is based, the risk of toxicity may be significant. In the case of mirex, an exposure at the RfD of 2 x 10^{-6} mg/kg/day is 10,000-fold below the level at which adverse effects have been observed in the vole, the experimental species found to be most sensitive to the effects of mirex.

] safety factors incorp.

Many of the pathways examined in this risk assessment have HI values less than the target HI of one. These include dermal contact with soil by individuals whose residences are in the flood plain, incidental ingestion of creek sediment and dermal contact with sediment by local residents who visit the creek for recreational activities, and ingestion of game by local hunters. The HI associated with incidental ingestion of soil by flood plain residents is approximately 3, only slightly higher than the target HI of one.

Several of the indirect exposure pathways evaluated in the baseline assessment of current and future use conditions associated with the MFLBC exceed the target HI of one: ingestion of vegetables by flood plain residents, ingestion of fish from the MFLBC, and ingestion of beef and milk by local farmers under the assumption that there are no fences to restrict access of livestock to the MFLBC. In the current use scenario, none of the HI values

are exceeded by more than 15 fold. In the future use scenario, the highest HI of 167 is for consumption of fish from the section of the MFLBC upstream of Lisbon Dam under the assumption that no fishing advisory is in place. The conservatively estimated potential exposure levels upon which these HI values are based are still far below the exposure level shown to cause any demonstrable toxicity in the most sensitive species.

D. Uncertainties and Limitations in the Risk Assessment Process

Risk assessment provides a systematic means for organizing, analyzing, and presenting information on the nature and magnitude of risks posed by chemical exposures. Nevertheless, uncertainties and limitations are present in all risk assessments because of the quality of available data and the need to make assumptions and develop inferences based on incomplete information about existing conditions and future circumstances. These uncertainties and limitations should be recognized and considered when discussing quantitative risk estimates.

In general, the uncertainties and limitations in the risk assessment can be classified in the following categories:

- environmental sampling and laboratory measurement;
- mathematical fate and transport modeling;
- receptor exposure assessment; and
- toxicological assessment.

1. Uncertainties in Environmental Sampling and Laboratory Measurement

The quality of the analytical data used in a risk assessment depends on the adequacy of the set of rules or procedures that specify how a sample is selected and handled, i.e., the sampling plan (USEPA 1988a). Uncertainties that may be associated with the data include sampling errors, laboratory analysis errors, and data analysis errors. The quality assurance and quality control review procedures used to minimize these uncertainties are described in other parts of the RI.

2. Uncertainties in Mathematical Fate and Transport Modeling

The use of mathematical models to predict the fate and transport of chemicals is well accepted in the professional engineering community and is endorsed by USEPA

in its Superfund Exposure Assessment Manual (USEPA 1988b). USEPA does not, however, provide specific guidance concerning the selection of specific models from among a wide variety available for a given purpose. Indeed, the trade-off between simplicity, generality, and accuracy is best made by considering the needs and available data of the site in question.

Because few environmental models have been authoritatively verified by field observations, there is some uncertainty associated with the use of a mathematical model for predicting environmental quality. In general, the selected models have been developed by contractors to USEPA or have been used by USEPA regulators and scientists for assessments similar to the one conducted here. In accordance with USEPA guidelines, conservative assumptions have been generally made in an effort to overestimate rather than underestimate risk.

3. Exposure Assessment Uncertainties

In any risk assessment, a large number of assumptions must be made to assess potential human exposure. In the conduct of the exposure assessment, it was necessary to develop assumptions about general characteristics and potential patterns of human exposure of the population in the vicinity of the MFLBC. In developing exposure scenarios, the upper bound of reasonably foreseeable circumstances was used to model risks based on guidelines from the USEPA. In developing the future use scenarios, exposure assumptions were made that assumed the absence of actions already taken to mitigate exposures to chemicals of potential concern in the MFLBC. Specifically, hypothetical future exposures were estimated assuming the lack of a fishing advisory above Lisbon Dam and the absence of fences installed to prevent access of livestock to the MFLBC and its floodplain.

For each exposure pathway modeled, assumptions were made about the number of times per year an activity could occur, the routes of exposure by which an individual could be exposed, the amount of contaminated media to which an individual could be exposed by the activity, and the amount of chemical that could be absorbed by each route of exposure. In absence of site-specific data, the assumptions used in this baseline risk assessment are generally those consistent with USEPA guidance for deriving estimates of the reasonable maximum exposure (RME, defined by USEPA as "the maximum exposure that is reasonably expected to occur at a site" (USEPA 1989a)). Many of the exposure variables recommended by USEPA for the RME case

represent 90th to 95th percentile values. When several upper bound values (i.e., 90th or 95th percentile values) are combined in estimating exposure for any one pathway, resulting risk estimates may well be in excess of the 99th percentile exposure and thereby outside the range of exposures that might reasonably be expected to occur at a site. To the extent possible, an attempt was made to avoid combining too many upper bound exposure variables in assessing any pathway. Because information on the statistical distribution of exposure variable values is limited, however, it is not always possible to characterize exposure estimates quantitatively.

4. Toxicological Assessment Uncertainties

In the great majority of risk assessments, as in the current risk assessment, available scientific information is insufficient to provide a thorough understanding of all the toxic properties of chemicals to which humans are potentially exposed. It is generally necessary, therefore, to infer these properties by extrapolating them from data obtained under other conditions of exposure, generally in laboratory animals.

Experimental animal data have been relied upon for many years by regulatory agencies and other expert groups for assessing the hazards and safety of human exposure to chemicals. This reliance has been supported in general by empirical observations. There may be differences in chemical absorption, metabolism, excretion, and toxic response, however, between humans and the species for which experimental toxicity data are generally available. Uncertainties in using animal data to predict potential effects in humans are introduced when routes of exposure in animal studies differ from human exposure routes; when the exposures in animal studies are short-term or subchronic; and when effects seen at relatively high exposure levels in animal studies are used to predict effects at the much lower exposure levels found in the environment. The methods for dealing with these uncertainties in the toxicological assessments for noncarcinogens and carcinogens is discussed below.

a. Uncertainties in the Characterization of the Toxicity of Noncarcinogens

In order to adjust for uncertainties such as those discussed above, regulatory agencies often base the acceptable daily intake (or for USEPA, the RfD) for noncarcinogenic effects on the most sensitive animal species, i.e., the species which experiences adverse effects at the lowest dose. This dose is then

adjusted via the use of safety factors or uncertainty factors in order to compensate for lack of knowledge regarding interspecies extrapolation and to guard against the possibility that humans are more sensitive than the most sensitive experimental animal species tested.

This risk assessment has used USEPA's RfD for mirex derived in this manner. In the case of mirex, the USEPA based the RfD for the compound's noncarcinogenic effects on the most sensitive species tested (the vole) and divided the lowest level shown to cause adverse effects in this species by an uncertainty factor of 10,000.

b. Uncertainties in the Characterization of the Toxicity of Carcinogens

For many substances that are carcinogenic in animals, there is uncertainty as to whether they are also carcinogenic in humans. While many substances are carcinogenic in one or more animal species, only a small number of substances are known to be human carcinogens. The fact that some chemicals are carcinogenic in some animals but not in others raises the possibility that not all animal carcinogens are human carcinogens. Regulatory agencies generally assume that humans are as sensitive to carcinogens as the most sensitive animal species. This is a policy decision designed to prevent underestimating risk, but it introduces considerable uncertainty, i.e., a greater probability of overestimation.

In addition, there are several mathematical models available to derive low-dose SFs from high exposure levels used in experiments. The model used by USEPA (and therefore in this risk assessment) is the linearized multistage model, which provides a conservative estimate of risk at low doses (i.e., the model is likely to overestimate the actual SF). Several of the alternative models often predict lower risk at low doses, sometimes by orders of magnitude. Thus, the use of the linearized multistage model ensures a conservative estimate of the SF. The lack of knowledge regarding the validity and accuracy of this model, however, contributes to the uncertainties in cancer risk estimates.

For suspected carcinogens, the normal procedure used by regulatory agencies, and therefore used here for mirex, is to use the 95% upper confidence limit estimated by the linearized multistage model. Use of the 95%

upper confidence limit value rather than the SF that represents the maximum likelihood estimate provides an estimate of the upper bound on risk.

One step in the methodology used by USEPA to estimate an SF from the animal data involves interspecies scaling (i.e., extrapolation) of dose from laboratory animals to humans in order to compensate for differences in such factors as size, lifespan, and basal metabolic rate. The USEPA extrapolates doses on the basis of milligrams of chemical per square meter body surface area per day ($mg/m^2/day$). Doses are also often extrapolated between species on a body weight basis, i.e., milligrams of chemical per kilogram of body weight per day (mg/kg/day). Both scaling factors have been used in risk assessments by different federal agencies. A number of scientists have concluded that mg/kg/day is the unit of dosage measurement giving the closest correlation between species (Allen et al. 1987; Crump et al. 1989). The surface scaling factor gives higher risk estimates per unit of dose than does the body weight scaling factor. For mirex, if body weight extrapolation is applied to the animal data used by USEPA, it would result in a potency factor approximately six-times smaller, or 0.31 $(mg/kg/day)^{-1}$ (USEPA 1987). This value still represents a 95% upperbound estimate of the carcinogenic potency of mirex at low doses. Use of this SF rather than the one developed by USEPA would result in risk estimates approximately six times smaller than those estimated in this assessment.

Application of these mathematical low-dose extrapolation models for carcinogens is predicated on the conservative assumption generally made by regulatory agencies that no threshold exists for carcinogens, i.e., that there is some risk of cancer at all exposure levels above zero.[7] As previously noted, this no-threshold hypothesis for carcinogens is by no means proven, and may not hold for some carcinogens that do not appear to act directly on genetic material (DNA). Mirex has been tested for potential to damage genetic material in a variety of mutagenicity assays and has been shown consistently to be negative for genotoxic potential (see Appendix B). Although the mechanism by which mirex causes an increased incidence of tumors in

[7] While this suggests that any exposure to a carcinogen poses some risk of cancer, the probability may be extraordinarily small, so that, for all practical purposes, no risk exists.

experimental animals is not known with certainty, the available genotoxicity data suggest that mirex does not directly interact with DNA. The apparent absence of genotoxicity of mirex raises the possibility that mirex may have a threshold for carcinogenicity. If this is the case for mirex, the risk assessment methods applied to mirex may substantially over-estimate low-dose cancer risk.

c. Uncertainties Introduced by Lack of Toxicity Information

In most risk assessments, chemicals are present that cannot be included in the quantitative risk assessment because little or no information on the toxicity of the chemical is available. One of the chemicals identified in the current assessment as potentially site related, diphenyl sulfone, was not carried through the assessment because the available toxicity data were limited to an LD_{50} of 320 mg/kg in the rat (NIOSH 1990). An LD_{50} is the single dose calculated to be lethal to 50 percent of the animals, and is generally not considered a sufficient basis for evaluating the toxicity of a chemical associated with chronic low-dose exposures. Empirical studies of acute and chronic toxicity data for other chemicals, however, have shown that chronic no-observed-effect levels (NOELS) can be approximated from LD_{50} values by dividing the LD_{50} values by factors ranging from about 1,000 to 3,120 (McNamara 1976; ENVIRON 1985; Layton et al. 1987). This procedure does not result in an accurate prediction of the chronic NOEL, but is unlikely to underestimate the chronic NOEL for any given chemical. An RfD is generally derived from a chronic animal study by dividing the chronic NOEL by an uncertainty factor of 100. Thus, a factor of about 100,000 can be applied to an LD_{50} value to approximate an exposure level that is unlikely to underestimate the RfD.

Thus, a lower-bound on the RfD for diphenyl sulfone can be estimated as 3.2×10^{-3} mg/kg/day by dividing the reported LD_{50} for diphenyl sulfone of 320 mg/kg by 100,000. When this value is used in conjunction with the maximum detected concentration in the toxicity-concentration screen for chemicals in sediment as presented in Chapter III, the relative risks from diphenyl sulfone are shown to be very small. Any potential risks posed by diphenyl sulfone would likely to be orders of magnitude smaller than those chemicals identified as chemicals of potential concern in this assessment.

IX. COMPARISON OF ENVIRONMENTAL CONCENTRATIONS OF CHEMICALS OF POTENTIAL CONCERN TO ARARS or Remedial Response Levels

A. Introduction

According to the Superfund Amendments and Reauthorization Act (SARA), applicable or relevant and appropriate requirements (ARARs) must be considered in the development of alternatives for remedial action at Superfund sites. ARARs are promulgated regulations which "are of general applicability and are legally enforceable" (52 FR 32498). There are three general types of ARARs: chemical-specific, location-specific, and activity-specific. Only chemical-specific ARARs are considered in public health risk assessments.

Chemical-specific ARARs are usually health-based or risk-based numerical values or methodologies that, when applied to site-specific conditions, result in the establishment of numerical values (USEPA 1988c). Chemical-specific ARARs typically include the Safe Drinking Water Act Maximum Contaminant Levels (MCLs) or MCL Goals (MCLGs); the Clean Water Act Water Quality Criteria (WQC); and the Clean Air Act National Ambient Air Quality Standards (NAAQS) or National Emissions Standards for Hazardous Air Pollutants (NESHAPS). Of the above, the only potential ARAR identified for a chemical of potential concern is the USEPA water quality criterion for mirex of 0.000001 mg/l for freshwater and marine aquatic life. As noted in previous chapters of this report, however, mirex was not detected in any surface water sample.

As presented in previous chapters of this report, concentrations of the chemicals of potential concern were identified in flood plain soils, sediment, fish tissue, and game. In addition, concentrations of chemicals of potential concern were modeled for vegetables, beef, and milk. In this chapter, potential chemical-specific ARARs are identified and compared to the chemical concentrations found or modeled in these media.

B. Identification of Potential ARARS

No potential chemical-specific ARARs were identified for the flood plain soils, sediment, game, vegetables, beef and milk. The only potential ARAR identified for this baseline risk assessment is the action level for mirex in the edible portion of fish of 0.1 ppm established under the Federal Food, Drug, and Cosmetic Act (FFDCA) (21 U.S.C. 321 et seq.). This level represents the pesticide residue limit at or above which the Food and Drug Administration (FDA) will take action to remove products from the market.

C. Comparison of Environmental Concentrations of Site Chemicals of Potential Concern to ARARs

The fish fillet samples exceeding the action level for mirex are listed in Table 35. Only fish sampled above the Lisbon Dam were found to contain concentrations of mirex at levels greater than the action level.

TABLE 35 Fish Fillet Samples Exceeding Mirex Action Level of 0.1 ppm[1]	
Sample Identification	**Sample Concentration (ppm)**
F1-08-UT	0.436J
F1-09-UT	3.48J
F1-13-UT	1.82J
F1-15-UT	0.207
F1-18-UT	0.195J
F1-23-UT	1.29
F1-28-UT	0.26
[1] Action level applies to edible portion of fish.	

X. ENVIRONMENTAL RISK ASSESSMENT

A. Introduction

The objective of this environmental risk assessment is to characterize the potential risks to ecological resources from hazardous substances that may have migrated from the Site to the MFLBC. [redacted] chemical exposure. Therefore, this assessment considers the measured exposure, plus the available guidance on fate and toxicity of the selected compounds with regard to the known habitats and likely species in the area.

Based on the site characterization analytical data, past operations at the Site, and the availability of ecotoxicological effects thresholds for hazardous substances, [redacted] The analytical data on chemical substance levels in surface water, sediments, flood plain soils, and fish, and the station descriptions plus field observations on habitats and fish and wildlife species are detailed in the RI (ERM-Midwest, Inc. 1991).

This assessment basically is consistent with the guidance contained in the USEPA environmental risk assessment manual entitled "Risk Assessment Guidance for Superfund, Volume II - Environmental Evaluation Manual" (USEPA 1989b). Additional guidance with regard to ecotoxicological thresholds/criteria for fish and wildlife species is taken from USEPA, U.S. Fish & Wildlife Service, and other published scientific reports.

The environmental risk assessment is divided into seven sections as follows:

Section A - Introduction (covering assessment objectives and scope)
Section B - MFLBC Sampling Program Summary
Section C - Ecological Resource Characterization
Section D - Site Related Chemicals of Potential Concern
Section E - Exposure Characterization
Section F - Toxicity Thresholds for Mirex
Section G - Risk Characterization

B. MFLBC Sampling Program Summary

The most recent MFLBC sampling program was conducted by ERM-Midwest, Inc. and is summarized in the RI (ERM-Midwest, Inc. 1991). A total of 22 surface water, 56 fish tissue, and 54 sediment or flood plain soil samples were collected from Slanker Pond and locations along the MFLBC from upstream of the Site to near East Liverpool, Ohio. A total of 52 stations were sampled between April 16 and May 21, 1990. Station #1 is in the MFLBC, upstream of the Salem municipal wastewater treatment plant (WWTP) outfall. Stations #2 to #5 are downstream of the WWTP but upstream of where Feeder Creek flows into the MFLBC. Station #6 includes Slanker Pond, which is between the Site and the MFLBC. Stations #7 through #39 are in the MFLBC and above the Lisbon Dam with the exception of Station #29, which is in the Stone Mill Run tributary to MFLBC. Stations #10, #12, #17, #19 and #27 also included flood plain samples associated with farms or swampy areas. Stations #40 through #52 are in the MFLBC downstream of the Lisbon Dam, with the exception of Station #47, which is in the West Fork LBC, and Station #50, which is in the North Fork LBC.

what is the significance of this?

why were these sampling spots chosen -- what are they likely to represent?

C. Ecological Resource Characterization

1. Habitat

ERM-Midwest, Inc.'s detailed summary of station descriptions and field observations of habitat for the 52 stations along the MFLBC is presented in the RI. In general, the terrestrial habitat goes from a "scrub/shrub wetland fringe" at station #1 into forested land at stations #2 to #5. Station #6 includes Slanker Pond. Most of the remaining stations above the Lisbon Dam are forested or forest-wetland with the exception of two swamp or emergent-wetland areas, stations #25 and #33 to #34. Below the dam, i.e., stations #40 to #52, much of the habitat is forested.

what section?

The MFLBC itself is a series of shallow riffles and pools with a maximum depth less than 1 meter and with half of the stations less than 0.3 meters in depth. Stream width ranges from approximately 4-8 meters above the dam, to 15-35 meters below. Average stream velocity at the time of sampling was less than 0.5 meters per second, with a discharge of 5-40 cubic feet per second (cfs) above the dam, and 100-300 cfs below. The creek substrate varied from bedrock outcrops and rubble-gravel-boulders in some areas, to sand, silt and clay in the forested wetlands and swampy areas. Water quality parameters including temperature (8-16° C), dissolved oxygen

(8-16 mg/l), specific conductance (150-600 umhos/cm) and pH (7-9) indicated generally good water quality.

2. Species

No attempt was made to enumerate the numbers of species or individuals present, or to assess any population or community parameters as indicators of ecosystem health/condition. Observations made during sampling did, however, confirm the presence of numerous biota in the areas adjacent to the 52 stations (ERM-Midwest, Inc.'s list of species at each station is included in the RI).

The mixed hardwood forests, shrubs and grasses are those common to the midwest. Wildlife were observed at nearly all of the sites and included: raccoon, beavers, muskrats, mallard ducks, wood ducks, Canada geese, great blue herons, kingfishers, sandpipers, pheasants, various songbirds, woodpeckers, watersnakes, tadpoles, frogs and snapping turtles. Horses, donkeys and deer were seen below the dam. Twelve families and over 50 species of fish were collected for tissue samples. Herbivores, omnivores and carnivores were represented at nearly all the sites.

An indication of general stream quality appeared in the USEPA 1987 survey of benthos in Little Beaver Creek (Metcalf & Eddy 1988). Kick net sampling indicated relatively good overall stream quality in the MFLBC. The most obvious pattern in the data was a higher number of pollution tolerant species (e.g., Tubificidae [worms] and Chironomidae [midges]) for the first approximately three river miles below the Salem municipal treatment plant, and then replaced by a higher number of pollution sensitive species (e.g., Tricoptera [caddisflies] and Ephemeroptera [mayflies]) downstream. This pattern could be attributed to solids and nutrients associated with treatment plant effluents.

Again, ERM-Midwest, Inc. did not attempt to do a thorough search for biota. However, none of the fish, birds or small mammals that were identified appear on the Ohio Endangered and Threatened Species List. The Ohio Natural Heritage Program Data Service shows no record of threatened or endangered fish or fish-eating wildlife species in the Little Beaver Creek watershed (the Heritage Program Inventory search was conducted in April 1991).

D. Chemicals of Potential Concern

Mirex (and photomirex) is the only potentially site-related chemical of concern that: (1) was detected with significant frequency (i.e., >5% of samples, USEPA 1989a) in MFLBC water, sediment or fish, and (2) for which there are available ecotoxicology data and guidance for the protection of fish and wildlife.

Mirex was detected in 34 of 54 sediment samples and 53 of 56 fish samples at detection limits of 0.018 and 0.025 mg/kg in sediments and fish tissues, respectively. No mirex was detected in surface water samples at a detection limit of 0.000005 mg/l (5 parts per trillion). Analytical results are presented in the RI.

1. Mirex Properties and Status

Empirical formula:	$C_{10}\,Cl_{12}$
Molecular weight:	545.5
Water solubility:	1 ppb (freshwater)
Henry's Law Constant:	5.16×10^{-4} atm-cu m/mol
Koc:	$2.4 \times 10^{+7}$

Mirex (perchloropentacyclodecane) has been used extensively in pesticidal formulations to control the imported fire ant and as a flame retardant in electronic components, plastics and fabrics. In 1978, the USEPA banned the use of mirex as a pesticide, partly because of the hazard it imposed on non-target biota. These included: delayed mortality in aquatic and terrestrial fauna, adverse effects on reproduction, early growth and development, plus high bioaccumulation and biomagnification in the food chain (Eisler 1985).

2. Mirex Fate

Mirex is a very stable and persistent organochlorine compound, being resistant to chemical, photolytic, microbial and thermal degradation. There is evidence, however, for some degradation to monohydro- (photomirex) and dihydro-derivatives, which are about as biologically active as mirex (Eisler 1985). Mirex adsorbs very little UV light in the environmentally relevant range of > 290 nm. A photodegradation experiment conducted in pure water for six months showed a half-life (i.e., time required for half of the starting material to be lost) of about 1 year (Smith 1978).

Mirex has a low solubility in water, not exceeding 1 ppb in freshwater and 0.2 ppb in seawater. It is highly soluble in fat and accumulates in fatty tissue. Mirex is rapidly adsorbed onto various organic particles in the water column, including algae, and eventually is removed to the sediments. With its relatively high Koc value of 2.4 x 10^{+7}, mirex will strongly adsorb to organic materials in soil and will be immobile except for movement via erosion to surface waters (Smith 1978). Not surprisingly, mirex is persistent in terrestrial and aquatic soils/sediments. Degradation half-life estimates range to 10 years or more. In biological systems, the elimination half-lives range from 30 days in quail to 130 days in fish, and to more than 10 months in rats (Eisler 1985).

Bioaccumulation of mirex is high in aquatic organisms, with bioconcentration factors (BCF) in the thousands for algae and aquatic invertebrates, and up to tens of thousands or more for fish. A log BCF of 7 was calculated for mirex based on Lake Ontario rainbow trout tissue samples versus the average level in the lake water (Oliver and Niimi 1985). BCFs from feeding mirex to birds and mammals are generally less than 50, with the highest levels in fat tissue and in eggs. The significance of mirex residues in various tissues is unresolved, as is the exact mode of action of mirex and its metabolites. A concern is that for animals at the top of the food chain, toxic levels may be reached before equilibrium levels are reached.

3. Mirex Toxicity (non-human)

96-hour LC50s were not obtained at the highest levels tested for aquatic insects, daphnids and fish; however delayed mortality frequently occurred after extended periods of exposure (Eisler 1985). This delay is presumably because it takes some time for mirex to get into the organisms and accumulate to toxic levels. Significant delayed mortality was observed for freshwater and estuarine crustaceans (i.e., crayfish and shrimp) after exposures as low as 0.0001 mg/l in the water (USEPA 1986c). The maximum acceptable toxicant concentration (MATC) determined for sublethal effects is less than 0.0024 mg/l for amphipods based on growth inhibition, less than 0.005 mg/l for bluegills based on growth, 0.034 mg/l for fathead minnows based on impaired reproduction, and greater than 0.034 mg/l for daphnids and midge based on reproduction and on emergence, respectively. Numerous other sublethal effects have been observed for algae, invertebrate and fish species (USEPA 1986c).

Birds appear comparatively resistant to mirex. Most investigators agree that comparatively high dietary concentrations of mirex had little effect on growth, survival, reproduction and behavior of nonraptors, including chickens, mallards, quail and red-winged blackbirds. It is possible that, at the top of the foodchain, raptors could ingest enough mirex-containing food organisms to affect reproduction (Eisler 1985). Mortality due to dietary mirex can occur, although high death rates were usually associated with high dietary concentrations and long exposure periods (Eisler 1985). For example, 27% of mallard ducks died when exposed to 100 mg/kg mirex in the diet for 25 weeks, 50% of ring-necked pheasants died when exposed to 1500 mg/kg in the diet for 5 days, and 20% of Japanese quail died in 5 days when exposed to 5000 mg/kg mirex in the diet.

Significant mortality was noted when voles, mice and rats were fed dietary levels of mirex in the 1-10 mg/kg range for 13 to 60 weeks. The no-effect level based on mortality for prairie voles is 5 mg/kg mirex in the diet, and less than 1.8 mg/kg mirex in the diet for old field mice (Newell 1987). Decreases in young survival have been observed when mirex at levels of 5 mg/kg mirex in the diet is fed to breeding rats. Effects have also been observed on reproductive performance and behavioral development in the prairie vole at intake levels of the adult animals as low as 0.1 mg/kg (Eisler 1985).

E. Exposure Characterization

A total of 52 stations were sampled for mirex and photomirex in surface water, sediments or flood plain soil, and fish between April 16 and May 21, 1990. A complete list of sample stations, sample types and chemical levels appears in the RI.

No mirex or photomirex was detected in any of the surface water samples (d[redacted]l). Whole body fish tissue levels of mirex were below 0.1 mg/kg at stations #1 (upstream of the POTW), in the ponds, in the stations just upstream of the Lisbon Dam, and in all stations downstream of the dam. The highest whole body fish tissue levels (i.e., between 1.0 and 6.2 mg/kg) were at stations #9, 13, 22 and 23. The remainder of the stations (i.e., #s 5, 7, 8, 15-20, and 28-29) had fish tissue levels generally between 0.1 and 0.5 mg/kg mirex. Small amounts of photomirex were found in fish tissues; however, photomirex (which is assumed to have the same toxicity as mirex) did not exceed 10% of the mirex levels. Photomirex was therefore not added to mirex levels for the purpose of the exposure characterization. ← Should be added in additive exposure = No Risk

Sediment or flood plain soil samples were taken at all but two stations. The level of mirex in sediments at station #1 was less than 0.010 mg/kg. Stations #10, 12 and 14 had the highest sediment levels which were 1.68, 2.82 and 1.20 mg/kg, respectively. Stations #11 and #13 had approximately 0.5 mg/kg mirex. With only a few exceptions, stations #15 to #30 had sediment levels between approximately 0.05 and approximately 0.15 mg/kg, and stations #31 to #52 were less than 0.050 mg/kg down to non-detect. Only a few samples had measurable levels of photomirex and these levels were typically less than 1% of the mirex levels. Photomirex was therefore not added to mirex for the assessment.

should be added in to mirex: additive Risk

Flood plain stations #10, #12, #17 and #27 had higher mirex levels (3.04, 4.54, 1.57 and 0.71 mg/kg, respectively) than did MFLBC sediments at the same stations (1.68, 2.82, 0.08, and 0.16 mg/kg, respectively). Further downstream the flood plain soil samples were comparable to sediments, i.e., in the 0.01-0.15 mg/kg range.

These data are consistent with the results of previous investigations. Samples of fish and sediment taken by OEPA and USEPA in 1986 and 1987, respectively, indicated mirex exposure beginning in the MFLBC in the vicinity of the Site and extending downstream to the Lisbon Dam. Mirex concentrations were highest near the Site, declining downstream but then increasing again where stream sediment settles in Egypt Swamp (north of Lisbon). Fourteen of 20 fish samples taken above Lisbon in 1987 had mirex concentrations exceeding 0.1 mg/kg. Although some fish containing mirex were captured downstream of the Lisbon Dam, sediments below the dam and downstream to the Ohio River did not contain detectable levels of mirex.

F. Toxicity Thresholds for Mirex

In addition to the ecological toxicology data presented in Section D.3, there are several published reviews which systematically evaluate the available data and offer conclusions regarding toxicity thresholds (or safe levels) for mirex. Thresholds have been estimated for exposures from water, sediments and edible tissues.

1. Water

[redacted]. This criterion is based upon an application factor of 0.01 (or 1%) applied to the lowest level at which effects were observed on several crustacean species. The lowest effect level was 0.0001 mg/l mirex for the most sensitive species tested.

This is Below The Det. Level used for Mirex in Surface Water

2. Sediment

Newell and coworkers (1989) have applied the proposed USEPA sediment quality criteria methodology (USEPA 1989c) to mirex along with many other sorptive chemicals. The bioavailable fraction of nonpolar organics, like mirex, has been correlated with the organic carbon content of the sediments. Adsorptivity is the controlling mechanism and the sediment pore water concentration is the bioavailable fraction. Using the USEPA's guidelines, the mirex effects threshold would be approximately 0.037 mg/kg in a sediment with a 1% organic carbon content, 0.111 mg/kg at 3% organic carbon, etc. These thresholds are wildlife residue-based; that is, sediment exposure above the threshold would be predicted to result in accumulation of mirex through the aquatic food chain to toxic levels in wildlife that eat aquatic organisms.

3. Fish Tissue

Newell and coworkers (1987) have also conducted an extensive review of the toxicity of mirex and other organochlorine chemicals to fish-eating birds and mammals. Mirex has been relatively well studied and there are considerable data on both acute and chronic effects on laboratory animals. A model was developed to predict toxicity thresholds for either birds or mammals that would be exposed to a chemical in 100% of its diet. The model is based on an average size (1 kg) animal and a food consumption rate of 0.15 kg/day for the mammal or 0.2 kg/day for the bird. Using the lowest chronic no observed effect level for rats, prairie voles, field mice, and mallard ducks, a toxicity threshold of 0.33 mg/kg was estimated for a fish-eating animal exposed to mirex. (The ducks, and birds in general, are less sensitive than are the mammals.)

Exceedence of this fish-flesh threshold would suggest that the potential exists for toxic effects in wildlife. Actual occurrence of effects would depend on the extent to which individual animals consume food with residues exceeding the threshold and the duration of consumption/exposure. Newell acknowledges the degree of conservativism in deriving the threshold values and suggests that exceedence of the fish flesh value by a factor of 10 would be expected to carry a "high risk" resulting in a specific adverse effect; again subject to the extent and duration of the exposure.

The fish-flesh threshold approach is preferable to the sediment-based wildlife approach since fish tissue levels are available for the chemical of interest. This is because the sediment-based approach relies on estimates of accumulation, where the actual measured tissue levels can be used in the fish-flesh threshold approach.

A toxic threshold for mirex should probably be expressed as "mirex and its degradation products" (Eisler 1985). But again, the measured levels of photomirex in MFLBC were a very small fraction of mirex levels and the conclusions of the assessment would not change by adding the two chemicals.

could if bioaccum is an important action here

G. Risk Characterization

For ecological risks, the ecotoxicity information and exposure data are integrated to address the likelihood that adverse effects may occur. The available data for the MFLBC, combined with the information and guidelines available from the USEPA and other published literature, allow the use of a simple quotient or ratio approach for the assessment. That is, is there evidence that the exposure could exceed the estimated toxicity thresholds for water, sediments or edible tissues as part of the food chain. This does not establish the existence of actual ecological impacts. Rather, the quotient method of risk characterization is an attempt to address the questions of what exposure pathways exist, what receptor populations could be at risk, and what is the likely magnitude of the exposure and the severity and time frame of the potential ecological effects.

The estimates of environmental exposures for MFLBC aquatic species and for wildlife that feed on them are based on RME values as described in Chapter VI. The RMEs represent the upper 95th percentile confidence limit of the mean chemical concentration in the medium of interest. Both the sediment and the fish samples are grouped into those taken above and below the Lisbon Dam because there is an obvious difference in the levels of mirex in these two areas. It is reasonable to use the RME analysis for the ecological assessment considering that the key receptor species (i.e., fish-eating birds and small mammals) are mobile and likely range beyond "a single sampling station," and considering that the toxicity threshold values and the pattern of mirex toxicity are related to longer-term, chronic exposures. The RME values for mirex are presented in Table 15.

The other important aspect of exposure is the presence of sensitive species. Although unquantified, there do appear to be a number of fish-eating bird, small mammal and reptile species present in the MFLBC area. In addition to those species observed by ERM-Midwest, Inc. during the site characterization (i.e., raccoon, heron, kingfisher and snapping turtles),

the Ohio Department of Natural Resources, District 3 Office of the Division of Wildlife, indicated via phone that mink and migratory osprey are also present in the watershed. It can also be assumed, based on the diversity of fish species captured, that there is a significant benthic invertebrate community (i.e., fish food) associated with the MFLBC sediments. The Metcalf & Eddy study discussed in Section C.2. of this chapter describes the MFLBC benthos as sampled in 1987.

The animal toxicology of mirex is reviewed in Section D.3., and the best available guidance for safe exposure levels is presented in Section F.

Comparing the ratios of toxicity thresholds versus the RME, and considering the other available information, the following can be concluded regarding the environmental risk from mirex in MFLBC:

- There is a relatively low risk to aquatic organisms associated with mirex in the water column. All of the water column samples were below detection. Although the detection limit (0.000005 mg/l) was above the USEPA criterion value by a factor of 5, the USEPA criteria is already 100-fold below the lowest chronic effect level of the most sensitive species tested. This 100-fold factor is intended to assure an adequate safety margin considering the inherent uncertainties in extrapolating laboratory toxicity data to a field situation.

- The MFLBC benthic community appears to be in good condition (based on the Metcalf & Eddy 1987 field survey), especially three or more river miles below the Salem municipal WWTP. There was no apparent correlation of benthic community composition with mirex levels in sediments. In fact, some of the higher sediment levels (i.e., 0.1 mg/kg mirex) coincided with diverse benthic communities including some pollution sensitive indicator species (i.e., caddisflies and mayflies). The survey was not designed or intended to address a specific chemical impact, but it does offer an indication of general stream quality.

- Fish-eating wildlife in the MFLBC above the Lisbon Dam are at some risk, albeit low, of chronic toxicity from consuming mirex in fish flesh and possibly from other aquatic foodchain sources.

- The 1.48 mg/kg RME for mirex in MFLBC fish tissues (whole body) exceeds estimated fish flesh chronic toxicity threshold of 0.33 mg/kg by a factor of 4.5. Although it is difficult to accurately quantify the risk to wildlife, Newell (1987) does allow for a 10-fold exceedence of the threshold before a "high risk resulting in a specific adverse effect" is expected. Another conservative assumption in the 0.33 mg/kg value is that although it is based on toxicity to mammals, it applies equally to birds, when in fact birds appear to be less sensitive to mirex.

- Only a few of the fish-eating bird species (i.e., heron and kingfisher) and possibly the mink are likely to have a diet that comes close to consisting of 100% fish (or other aquatic foodchain organisms) from the MFLBC. The osprey are migratory and are only in the area for a couple weeks in the spring and fall. The ducks, raccoons, snapping turtles, etc. consume some fish along with other aquatic and non-aquatic organisms. A diet of 100% contaminated food above the RME level is an assumption of the risk characterization.

- Fish-eating wildlife in the MFLBC below the Lisbon Dam are at no risk of chronic toxicity as the mirex RME is 0.044 mg/kg, or one-tenth of the toxicity threshold.

In summary, this risk assessment is not based on a data set that was intended to establish the existence of actual ecological impacts, but rather to characterize the potential risks based on the available exposure, toxicological and ecological information. The primary exposure pathway for mirex is through the aquatic foodchain to fish-eating wildlife. The persistent nature of mirex and its presence in MFLBC sediments and flood plain soils suggest that exposure could continue for some time. There are a few resident wildlife species in the area (i.e., heron, kingfisher and mink) that have fish or other aquatic organisms as their major food source, and therefore would very likely be exposed to mirex. The calculated RME value for mirex in fish (whole body) above the Lisbon Dam exceeds the estimated mirex toxicity threshold by 4.5 fold indicating the potential, but low risk of chronic, sublethal effects in these species. The actual occurrence of effects would depend on the extent to which individual animals consume those fish and other organisms with residues in excess of

the thresholds and the duration for which that consumption is continuous. The residue data for sediments and fish suggest that the exposure, and therefore the risk, does not extend below the Lisbon Dam. Finally, the RI-related observations of relatively diverse benthos, fish and wildlife in the MFLBC vicinity indicate that there are no large-scale, readily apparent impacts of mirex on the MFLBC ecosystem.

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